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16. Abstract (Limit: 200 words)

This Position Document addresses the risks and benefits of pesticide products containing the subject active ingredient. The Agency has determined that the use of products containing the subject active ingredient may meet or exceed a risk criterion described in 40 CFR Part 154. Potential hazards will be examined further to determine the nature and extent of the risk, and considering the benefits of the subject active ingredient, whether such risks cause unreasonable adverse effects on the environment.

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ALACHLOR

SPECIAL REVIEW
TECHNICAL SUPPORT DOCUMENT

SEPTEMBER 1986

Office of Pesticide Programs
Office of Pesticides and Toxic Substances
U.S. Environmental Protection Agency
401 M Street SW.
Washington, DC 20460

ERRATA SHEET

Page	Entry
II-19	Delete "for sweet corn" in footnote 3.
II-23	•Under entry for Ontario, Canada, delete "Ontario" in reference citation, and replace with "Bradley, J.".
	•Add following reference under entry for Minnesota: "(Klaseus, T., Personal Communication, 1986)".
	•Add following reference under entry for North Carolina: "(Chellman, C., Personal Communication, 1986)".
II-24	•Add following reference under entry for Kansas: "(Eiden, C., Personal Communication, 1986)".
	•Add following reference under entry for Nebraska: "(Eiden, C., Personal Communication, 1986)".
II-25	•Under first entry for Ontario, Canada, under Table 8B, change Positive Samples/Total figure from 10/2 to 10/28.
	•Under Comments, delete "rural well water; 1979-1984 tests", and replace with "well waters from SW Ontario, June-July, 1984".
II-26	.Change reference to the number of private and observation wells affected from (78) to (97).
II-33	Replace Montana with Missouri.
II-70	Under Rebuttal Comment 6, replace "overestimated" with "underestimated".
IV-12	•Make following corrections to Section E.1.a "Cancel Use on Peas". [Deleted text is stricken out; new text is underlined].
	If the registrations of alachlor procucts for use on peas were continued without restriction (Option 1), the dietary risks would be ~~reduced by-9-percent~~ unchanged. Residues in processed or cooked peas were not estimated.

If the registrations of alachlor products for use on peas were cancelled (Option 3), all risks associated with consuming alachlor-treated peas would be eliminated and the total dietary risk reduced by 9 percent. The cancellation of alachlor use on peas would result in little impact to farmers.

Make following corrections to Section E.1.b. "Cancel Use on Dry Beans". [Deleted text is stricken out; new text is underlined].

If the registrations of alachlor products for use on dry beans were continued without restriction (Option 1), the dietary risks would be ~~reduced by 32 percent~~ unchanged.

If the registrations of alachlor products for use on dry beans were cancelled (Option 3), all risks associated with consuming alachlor treated dry beans would be eliminated and the total dietary risk reduced by 32 percent. The cancellation of alachlor use on dry beans would result in little impact on farmers.

IV-13 Make the following corrections to Section E.1.c. "Cancel Use on Lima Beans". [Deleted text is stricken out; new text is underlined].

If the registrations of alachlor products for use on lima beans were continued without restriction (Option 1), the dietary risks would be ~~reduced by 7 percent~~ unchanged.

If registrations of alachlor products for use on lima beans were cancelled (Option 3), all risks associated with consuming alachlor treated lima beans would be eliminated and total dietary risk reduced by 7 percent. The cancellation of alachlor use on lima beans would result in little impact to farmers based on the limited data available on this crop.

V-9 Correct reference to National Research Council (1977) as follows:

National Academy of Sciences (1977) Summary Report: Drinking Water and Health, a Report of the Safe Drinking Water Committee, Advisory Center on Toxicology, Assembly of Life Sciences, National Research Council.

V-10 Correct reference to Reinert, J.C., as follows:

Reinert, J.C.: Nielsen, A.P.: Lunchick, C.; Hernandez, O.; Mazzetta, D.M. (1986). The U.S. Environmental Protection Agency's Guidelines for Applicator Exposure Monitoring. Toxicology Letters 33, p. 183-191.

Executive Summary

On January 9, 1985, the Environmental Protection Agency issued a Notice of Initiation of Special Review of Registrations of Pesticide Products Containing Alachlor (50 FR 1115), and the Alachlor Position Document 1 (PD-1) that detailed the basis for the Special Review. Alachlor is the common name for 2-chloro-2' 6'diethyl-N-(methoxymethyl) acetanilide and is often referred to by its trade name, Lasso®.

The Special Review action was based on the Agency finding that pesticide products containing alachlor met or exceeded one of the risk criteria in 40 CFR 162.11(a)(3)(ii)(A), which provides that a Special Review shall be initiated if a pesticide, its metabolites, or degradation products "induces oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation or dermal exposure." The Special Review was initiated based on laboratory studies in rats and mice demonstrating the potential for alachlor to produce oncogenic effects in mammals.

The risk criteria set forth in 40 CFR 162.11(a)(3)(ii)(A), were superceded by new criteria set forth in 40 CFR 154.7. Alachlor would also meet the oncogenicity criterion in 40 CFR 154.7(a)(2) and thus, a Special Review would have been initiated even if the new criteria had been in effect when the alachlor Special Review began in 1985.

This document, the Alachlor Technical Support Document (TSD), further addresses the risks and benefits associated with the continued use of alachlor, considering new information that has been reviewed by the Agency since the PD-1, and presents the Agency's proposed regulatory decision for alachlor.

Alachlor is an acetanilide herbicide registered for control of many annual grassy weeds and certain annual broadleaf weeds before they emerge from the soil. Alachlor is registered for use on the following sites: corn (field, sweet, pop, seed), soybeans, peanuts, dry beans, lima beans (green), mung beans, green peas (processing), cotton, grain sorghum, sunflowers, and certain ornamental plants. Approximately 94 percent of the annual 80 to 84 million pounds of alachlor active ingredient used in the United States is on three sites: corn (63 percent), soybeans (28 percent), and peanuts (3 percent). There are nine federally registered alachlor products, of which three are mixtures.

In assessing the hazard associated with the use of alachlor, the Agency reviewed data demonstrating that administration of alachlor to laboratory animals induced statistically significant oncogenic responses at multiple sites and doses in two sexes in the rat (nasal turbinate, stomach, and thyroid) and at one site at one dose in one sex in the mouse (lung). No new oncogenicity studies have been submitted since the issuance of the PD-1. Therefore, the Agency concludes that alachlor is a demonstrated animal oncogen. Although there are no data available indicating that alachlor caused oncogenic effects in humans, the Agency's position is that alachlor should be viewed as having the potential to be a human oncogen. According to the Agency's classification scheme (EPA, 1986), alachlor is assigned to category B2, a probable human carcinogen.

Exposure to alachlor can occur while applying the pesticide to agricultural crops, harvesting the crops, and eating the foods containing residues of alachlor. Exposure can also occur through drinking water contaminated with alachlor residues.

Applicator Exposure

In the PD-1, applicator exposure was determined from studies where the amount of alachlor an applicator was exposed to was assessed by measuring residue deposits of the chemical on gloves or gauze pads (patch data). The patch data measured worker exposure to alachlor during aerial and ground applications of the emulsifiable concentrate (EC), and microencapsulated (ME) and granular formulations. The exposure estimates assumed 50 percent dermal absorption for the EC and 12 percent dermal absorption for the microencapsulated and granular formulations. The risk to commercial ground applicators, assuming 30 days per year of exposure for 40 years and 80 percent protection by clothing, was estimated to be 10^{-3}

In response to the Registration Standard that was issued near the time the Special Review was initiated and PD-1, the registrant submitted several studies relevant to applicator exposure: an applicator study in which levels of alachlor were measured in urine (biomonitoring study), a dermal penetration study which indicated a 24 percent rate of dermal absorption for the EC formulation in the monkey, and a monkey metabolism study which indicated that the urinary tract is the primary route of excretion. The Agency also evaluated published exposure studies in preparation for the PD-2/3. The exposure data based on these published studies and the Monsanto patch data were used to estimate the range of exposure during the application of alachlor.

New risk estimates were then calculated using the biomonitoring data, surrogate data and new dermal absorption studies. The Agency believes that biomonitoring data, if supported by adequate and appropriate metabolism studies, generally provide a better measure of actual dosage received in the body than patch data. Thus, considering these new data, the Agency estimated the following ranges of applicator exposure and risks for both mixing and loading and ground boom application.

Private Farmers	1 day per year	$4x10^{-5}$ to $4x10^{-7}$
Private Farmers	3 days per year	$1x10^{-4}$ to $1x10^{-6}$
Commercial Applicators	15 days per year	$6x10^{-4}$ to $6x10^{-6}$

Also, data received from the registrant and available in the published literature indicate that pilots are expected to receive less exposure per pound of active ingredient applied than ground boom applicators. Thus, aerial application, which was voluntarily removed from the label by the registrant prior to the initiation of the Special Review, appears to result in less exposure to humans (provided mechanical flaggers are used) than ground boom applicators.

Dietary Exposure and Risk

In the PD-1 dietary exposure and risks were calculated for alachlor and one class of its metabolites, DEA. The total dietary exposure to the U.S. population was estimated using two different methods; first, by assuming that 100 percent of all crops were treated and that all crops contain residues of alachlor at the tolerance level, and second by using available information on residue levels actually measured in field tests. The exposure was estimated at 6×10^{-4} mg/kg/day and 4×10^{-4} mg/kg/day, respectively.

In response to the Registration Standard and PD-1, the registrant submitted actual residue data for alachlor and its two major classes of metabolites, DEA and HEEA, for the following crops and food sources: corn, soybeans, peanuts, meat, milk, poultry, and eggs. For the remaining crops (peas, beans, cottonseed, sorghum, and sunflower seeds), residue data were submitted for alachlor and one class of metabolites (DEA). The risks, utilizing these new data, assuming both 100 percent crop treated and a best estimate of the actual percent crop treated, are 2×10^{-5} and 2×10^{-6}, respectively.

Ground and Surface Water Exposure and Risk

In the PD-1, data were utilized that indicated alachlor was found in ground water at levels ranging from 0.01 to 16.6 ppb in three states and in Ontario, Canada. Data on surface water showed levels of alachlor contamination between 2 to 5 ppb.

In 1985, after the issuance of the PD-1, ground water sampling of 243 wells was completed. Data indicated that 237 wells contained no detectable levels of alachlor (< 0.2 ppb) with the concentrations in the six remaining wells ranging from 0.2 to 6.0 ppb. Also, the County and State agencies and the United States Geological Survey conducted ground water monitoring studies and found alachlor contamination in ground water in nine states sampled with concentrations ranging from 0.1 to 16.6 ppb (typical range of 0.2 to 2.0 ppb).

Surface water data on both raw and finished water were submitted in 1985 for 24 community water supplies (CWS). The results indicate that alachlor was detected in either raw or finished water at 14 locations with annualized mean concentrations ranging from < 0.2 to 1.5 ppb; no alachlor was detected at the remaining locations.

Benefits

The benefits of alachlor were assessed in terms of economic impacts which would result if the uses of alachlor were cancelled and users were forced to use the available alternatives. Cancellation of all uses of alachlor would be expected to result in first year losses at the farm level of $510 to $758 million, which represents both increased costs for weed control and decreased value of production. The overall benefits loss to society would range from about $302 to $508 million the first year. Such overall losses would be nominal to consumers of food products affected.

The economic impacts of the measures proposed by the Agency have been evaluated and have been determined to be minimal. The Agency is proposing to reclassify alachlor as a restricted use pesticide. Approximately 65 percent of all commercial and private farmers who use alachlor are already certified under state certification programs, according to data provided by the registrant. The Agency expects this percentage to be much higher due to EPA's recent requirement that users of corn root worm granular formulations be certified.

Private farmers would comprise the majority of the remaining uncertified group. The cost to each farmer to obtain certification would be minimal; some states do not charge a fee to private farmers for certification. The costs of additional certification and training attributable to this proposed measure would be borne by the states. EPA estimates that the states spend between $4 and $5 million annually on certification and training programs. The incremental cost attributable to the requirement that alachlor users be certified would be minimal.

Commercial applicators who use alachlor are most likely already certified due to their usage of other restricted use pesticides, and would incur no additional costs under this proposal.

The Agency has also determined that the impact of requiring the use of closed mixing/loading systems by applicators who treat 300 acres or more annually with alachlor is minimal. This requirement would affect approximately 40 percent of the farms using alachlor and it is assumed that most of the large farms already use closed mixing/loading systems, such as the shuttle system developed by Monsanto.

Proposed Regulatory Decision

In weighing the risks and benefits associated with the continued use of alachlor, the Agency reviewed a number of options to reduce exposure and risk.

The Agency proposes to allow the continued use of alachlor on agricultural and nonfood crops with certain conditions and label modifications to the registrations.

The Agency has determined that the upper bound U.S. dietary risk from alachlor residues on food crops is 2×10^{-6} based on the actual percent of crops treated with alachlor. The Agency believes this risk is reasonable given the benefits of continued use. The Agency's final regulatory decision on registrations of alachlor for use on peas, dry beans, and lima beans is being deferred, pending receipt of requested residue data for each of these commodities when they are processed. These data are required to be submitted by July 1987 pursuant to a 3(c)(2)(B) Notice issued to the registrant.

Based on the available data, the Agency has determined that the risks associated with alachlor exposure through ground water cannot be adequately assessed at this time. The true extent of the occurrence of alachlor in ground water is not known and cannot be properly estimated for most areas of the country. The Agency's position is that further monitoring will be necessary considering the large volume, multiregional use of alachlor, and the lack of statistically representative data from the available studies. Several ground water and soil leaching studies are being conducted by the registrant, various states, the U.S. Department of Agriculture, and the U.S. Geological Survey. The risk posed by alachlor from exposure through ground water will continue to be assessed along with the need for any regulatory action as new data become available.

Existing data on alachlor residues in surface water indicate that the risk from drinking water sources supplied by surface water will generally not exceed a range of 2×10^{-6} to 4×10^{-6}. The Agency believes this level of risk is reasonable given the benefits of continued use of alachlor products, and is not proposing regulatory action under FIFRA on alachlor residues in surface water. The Agency plans to promulgate regulations establishing a Maximum Contaminant Level (MCL) for alachlor under the Safe Drinking Water Act in the near future. These regulations would require the treatment of drinking water which contains alachlor residues in excess of the MCL, thereby maintaining the level of risk from exposure at reasonable levels.

The Agency is proposing the following conditions and label modifications to the alachlor registrations to reduce applicator risks to reasonable levels:

- The application of alachlor is restricted to use by certified applicators or persons under their direct supervision. The alachlor label must be amended to include the following statement:

  Restricted Use

  due to oncogenicity

  "For retail sale to and use only by Certified Applicators or persons under their direct supervision and only for those uses covered by the Certified Applicator's certification."

- The following tumor warning statement imposed in the alachlor Registration Standard must remain on the label:

  "The use of this product may be hazardous to your health. This product contains alachlor which has been determined to cause tumors in laboratory animals."

- The use of a closed mixing/loading system is required to be used by all mixer/loaders and/or applicators who treat 300 acres or more annually.

- Aerial application will be allowed to be reinstated on the alachlor label with the following additional label restriction:

  "Human flaggers prohibited. Aerial application may be performed using mechanical flaggers ONLY."

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I. INTRODUCTION

A. General Background and Organization

On January 9, 1985, EPA issued a Notice of Initiation of Special Review of Registrations of Pesticide Products Containing Alachlor (50 FR 1115), and the Alachlor Position Document 1 (PD-1) that detailed the basis for the Special Review. The Agency had determined that pesticide products containing alachlor met or exceeded the risk criterion in 40 CFR 162.11(a)(3)(ii)(A), which provides that a Special Review shall be initiated if a pesticide, its metabolites, or degradation products "induces oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation or dermal exposure." The alachlor Special Review was initiated based on laboratory studies in rats and mice demonstrating the potential for alachlor to produce oncogenic effects in mammals.

The risk criteria set forth in 40 CFR 162.11(a)(3)(ii)(A) were superceded by new criteria set forth in 40 CFR 154.7. Alachlor would also meet the oncogenicity criterion in 40 CFR 154.7(a)(2) and thus, a Special Review would have been initiated even if the new criteria had been in effect when the alachlor Special Review began in 1985.

The basis of EPA's decision to initiate the Special Review on alachlor is further detailed in a document entitled "Guidance for the Interim Registration of Pesticide Products containing Alachlor as a Active Ingredient" (Registration Standard) which was issued in November 1984 and in the PD-1. In the PD-1, EPA solicited comments on the risks and benefits associated with all the uses of alachlor.

This document, the Alachlor Technical Support Document (TSD), further addresses the risks and benefits associated with the continued use of alachlor, considers new information that the Agency has reviewed since the issuance of the PD-1, and presents the Agency's proposed regulatory decision for alachlor. The document addresses the risks and benefits of alachlor use and is comprised of five chapters. Chapter I is this introductory section. Chapter II evaluates the potential risks associated with alachlor use and includes descriptions and evaluations of the risk evidence and exposure data, the Agency risk assessments, and analyses of rebuttal comments regarding risk from alachlor use which were submitted in response to the Alachlor PD-1. Chapter III summarizes the economic benefits associated with alachlor use and the analysis of comments received on benefits. Chapter IV describes the possible regulatory options considered by the Agency, a risk/benefit assessment of these options, and a proposed regulatory decision for the various registrations of alachlor. Chapter V is the bibliography.

B. Legal Background

To obtain a registration for a pesticide under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended, an applicant for registration must show that the pesticide satisfies the statutory standard for registration. That standard requires, among other things, that the pesticide perform its intended function without causing "unreasonable adverse effects on the environment" (FIFRA section 3(c)(5)).

The term "unreasonable adverse effects on the environment" is defined (FIFRA section 2(bb)) as any "unreasonable risk to man or the environment, taking into account the economic, social and environmental costs and benefits of the use of any pesticide." To register a pesticide, the Administrator must weigh the risks of use against the benefits of each use.

The burden of proving that a pesticide satisfies the statutory standard for registration is on the proponents of registration and continues as long as the registration remains in effect. Under FIFRA section 6, the Administrator may cancel the registration of a pesticide or modify the terms and conditions of registration whenever it is determined that the pesticide causes unreasonable adverse effects on the environment. The Agency created the Special Review process (previously known as the RPAR process) to facilitate the identification of pesticide uses which may not satisfy the statutory standard for registration and to provide an informal procedure to gather and evaluate information about the risks and benefits of these uses.

The Special Review process, formerly called the Rebuttable Presumption Against Registration (RPAR), is a mechanism by which the Agency collects information on the risks and benefits associated with the uses of pesticides to determine whether any use causes unreasonable adverse effects to human health or the environment. The Special Review process is currently governed by 40 CFR Part 154.

Through the Special Review process the Agency (1) announces and describes the Agency's risk concerns regarding pesticidal use based on certain risk criteria, (2) establishes a public docket, (3) proposes a regulatory decision based on evaluation of the pesticide's risks and benefits associated with a pesticide's use.

Issuance of this Notice means that potential adverse effects and benefits associated with the use of pesticide products containing alachlor have been assessed and that the Agency has preliminarily determined that, unless the terms and conditions of registration are modified as proposed in this Notice, the risks from exposure to alachlor outweigh the benefits of its use.

In determining whether the use of a pesticide poses risks which are greater than the benefits, the Agency considers possible changes to the terms and conditions of registration which can reduce risks, and the impacts of such modifications on the benefits of use. If the Agency determines that such changes sufficiently reduce risks to the level where the benefits outweigh the risks, it may conclude the Special Review process. Unless the Agency determines that the basis of the Special Review has been rebutted, it will issue a Notice of Intent to Cancel pursuant to FIFRA section 6(b). This Notice of Intent to Cancel may specify changes in terms of the registration which the Agency considers sufficient to avoid cancellations.

C. Chemical Background

Alachlor is the common name for 2-chloro-2',6'-diethyl-N-(methoxymethyl) acetanilide. Alachlor is often referred to by its trade name, Lasso®.

1. Registered Uses and Production

Alachlor is produced in the United States by Monsanto Chemical Company. It has been registered since 1969 as a selective herbicide for control of many preemergent broadleaf weeds and grasses. Registered uses include selective weed control in cultivated agricultural land and woody ornamentals. Target species include barnyardgrass, crabgrass, foxtails, goosegrass, signalgrass, witchgrass, fall and Texas panicum, nightshade, carpetweed, pigweed, purslane and sandbur.

Alachlor is usually applied as a broadcast surface treatment by ground application dissolved in 15 or more gallons of water per acre. It may also be applied as a band treatment. The most commonly used formulations are the 43.5 and 45.1 percent emulsifiable concentrates (EC) and the 10 and 15 percent granular (G) products. A microencapsulated formulation (MicroTech, MT) was registered in 1983 for use on dry beans, lima beans, peas, potatoes, and soybeans. The alachlor EC formulations are generally applied as tank mixes with other herbicides. Alachlor also is sold in formulations mixed with either atrazine or glyphosate. Application rates vary from 1.5 to 4 pounds active ingredient per acre depending upon target species and use site.

According to EPA records, Monsanto is the only company holding Federal registrations for nine alachlor products. There are also nine intrastate registrations for alachlor which are marketed pursuant to 40 CFR 162.17.

2. Tolerances

Tolerances are maximum permissible residue levels established pursuant to the Federal Food, Drug and Cosmetic Act. Tolerances were established in 40 CFR 180.249 for residues of alachlor and its metabolites. Two major classes of alachlor metabolites have been identified, containing the diethylaniline (DEA) and hydroxyethylethylaniline (HEEA) moieties, resulting from the use of the herbicide in or on raw agricultural commodities. These tolerances are presented in Table 1.

3. Registration Standard and Special Review

The Pesticide Registration Standard and Guidance Document for Alachlor was issued in November 1984. A Registration Standard contains a thorough review of the scientific data base supporting a pesticide's registration and an identification of any data gaps in that data base. The Registration Standard may also give notice to registrants that additional data need to be developed. The Guidance Document stated that the Agency would not reregister any current products containing alachlor at that time; furthermore, the Guidance Document stated that alachlor was a candidate for Special Review.

In December 1984 the alachlor PD-1 was published in the Federal Register.

Table 1. Alachlor Tolerances

Commodity	Tolerance	Citation and Form
Beans, dry	0.1 ppm	(40 CFR 180.249 Alachlor and its metabolites calculated as alachlor)
Beans, forage	0.2 ppm	
Beans, lima (green)	0.1 ppm	
Cattle, fat	0.02 ppm	
Cattle, meat-by-products	"	" "
Cattle, meat	"	" "
Corn, fodder	0.2 ppm	" "
Corn, forage	"	
Corn, fresh, including kernels and corn with husks removed	0.05 ppm	" "
Corn, grain	0.2 ppm	" "
Cotton, forage	"	" "
Cottonseed	0.05 ppm	" "
Eggs	0.02 ppm	" "
Goats, fat	"	" "
Goats, meat-by-products	"	" "
Goats, meat	"	" "
Hogs, fat	"	" "
Hogs, meat-by-products	"	" "
Hogs, meat	"	" "
Horses, fat	"	" "
Horses, meat	"	" "
Milk	"	" "
Peanuts	0.05 ppm	" "
Peanuts, forage	3.0 ppm	" "
Peanut, hay	"	" "
Peanut, hulls	1.5 ppm	" "
Peas, forage	0.2 ppm	" "
Peas, hay	"	" "
Peas, pods removed	0.1 ppm	" "
Potatoes	"	" "
Poultry, fat	0.02 ppm	" "
Poultry, meat-by-products	"	" "
Poultry, meat	"	" "
Sheep, fat	0.02 ppm	" "
Sheep, meat-by-products	"	" "
Sheep, meat	"	" "
Sorghum, fodder	1.0 ppm	" "
Sorghum, forage	"	" "
Sorghum, grain (milo)	0.1 ppm	" "
Soybeans	0.2 ppm	" "
Soybean, forage	0.75 ppm	" "
Soybean, hay	0.2 ppm	" "
Sunflower seed	0.25 ppm	" "

II. RISK ASSESSMENT AND ANALYSIS OF PUBLIC COMMENTS

In the PD-1, the Agency assessed the potential risks related to the use of alachlor and the available information on its possible oncogenic and nononcogenic effects. The following risk assessment discussion restates the hazard evaluation presented in the PD-1 and includes a detailed discussion of the new information submitted in the areas of pharmacokinetics, metabolism and short term testing. The Agency's re-evaluation of the dietary, drinking water and applicator exposure and risks is also presented in this Chapter.

According to the Agency's Guidelines for Carcinogen Risk Assessment, the risk assessment process for a suspect carcinogen consists of four steps (EPA, 1986). In the first step, Hazard Identification, relevant information is presented and a qualitative weight-of-the evidence judgment is reached on the likelihood that the pesticide is a human carcinogen. In the second step, Dose-Response Assessment, experimental data are used in conjunction with certain assumptions and a mathematical model to extrapolate the likely upper bound of human cancer risk to the low dose range. The third step is Exposure Assessment in which human exposures via various routes and sources are estimated. Finally, in the fourth step, Risk Characterization, the results of the Exposure and Dose Response Assessments are coupled to project the plausible upper bound of the cancer risk under different conditions of exposures. This step also includes a summary of the strength of the qualitative evidence, plus a treatment of the uncertainties in the final assessment.

A. Hazard Identification (Qualitative Risk Assessment)

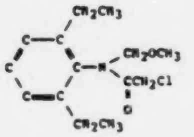
1. Physical-Chemical Properties and Exposure Pathways

Alachlor is the common name for 2-chloro-2',6'-diethyl-N-(methoxymethyl)acetanilide, CAS Number 15972-60-8, a preemergent herbicide. Other names for this pesticide include 2-chloro-N-(2,6-diethylphenyl)-N-(methoxymethyl)acetamide, CP 50144, and Lasso®. The Shaughnessy Code Number is 090501.

Alachlor has an empirical formula of $C_{14}H_{20}NO_2Cl$ and a molecular weight of 269.77. Table 2 lists the chemical and physical properties of alachlor. The structure of alachlor is shown in Figure 1.

Alachlor is registered for use as a pre-emergence herbicide on corn, soybeans, peanuts and other crops. Corn, soybeans and peanuts account for 94 percent of total alachlor usage. This use pattern and the environmental behavior of alachlor suggest that the principal pathways of human exposure are by direct contact during application, ingestion of commodities treated with alachlor, and through drinking water.

Figure 1.



Alachlor

Table 2. Physical and Chemical Properties of Alachlor[1]

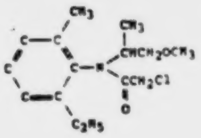
Melting Point	--	40 to 41°C
Specific Gravity	--	1.133 (25/15.6°C)
Solubility	--	Soluble in ether, acetone, benzene, alcohol (unspecified) and ethyl acetate; slightly soluble in hexane; solubility in water - 240 ppm
Octanol/Water Partition Coefficient	--	434
Stability	--	Stable (first detectable heat evolution at 105°C)
Appearance at room temperature	--	White, crystalline solid at 23°C

[1] Monsanto, 1973, 1979.

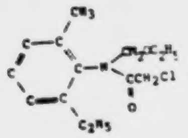
2. Structure-Activity Relationships

Alachlor is a substituted acetanilide. The Agency has concluded that at least two other chemicals in this class are oncogenic in laboratory animals. Metolachlor (2-chloro-2'-ethyl-6'-methylphenyl-N-(2-methoxy-1-methylethyl) acetanilide) has produced tumors in the rat liver (Ciba-Geigy, 1979). The structure of metolachlor is seen in Figure 2. Acetochlor when fed to CD-1 mice produced an increased incidence in combined adenomas and carcinomas of the lung at all doses (as well as other tumor types). Acetochlor also causes liver and thyroid follicular cell tumors in Sprague-Dawley rats. The structure of acetochlor is seen in Figure 2. Other structurally-related chemicals have either not been adequately tested or the review of testing has not yet been completed by the Agency. The pattern of oncogenic response appears to vary among the acetanilides.

Figure 2.



Metolachlor



Acetochlor

3. Metabolic and Pharmacokinetic Properties

In the PD-1, a metabolism study of alachlor in the rat was discussed (Monsanto, 1983a). The study indicated that alachlor is rapidly metabolized and eliminated as conjugates of mercapturic acid, glucuronic acid and sulfate in urine and feces (37.6 to 45.0% in urine and 47.7 to 51.1% in feces of males; 42.5 to 53.2% in urine and 37.0 to 49.3% in feces of females). Approximately 89 percent of the dosages were eliminated during the 10-day study, with most of the elimination occurring within the first 48 hours (half-life of 0.2 to 10.6 hours), followed by a slower phase (half-life of 5 to 16 days). Elimination as CO_2 was minimal.

Radioactivity from the administered dose was found in the blood and in the spleen, liver, kidney, and heart which may be a reflection of the amount of blood in those organs. In addition, a relatively high level of radioactivity was also found in the eyes, brain, stomach, and ovaries. Radiolabeled alachlor was found at some of the same sites where tumors occurred in the 2-year rodent feeding studies as discussed below which lend to the significance of tumor formation in test animals.

In response to the PD-1, Monsanto submitted several studies on the metabolism of alachlor in Rhesus monkeys and mechanistic studies on the binding of alachlor to blood components.

An intravenous metabolism study was conducted in Rhesus monkeys (Monsanto, 1984). In this study, alachlor was rapidly distributed in the blood within the first 15 minutes and rapidly eliminated in urine primarily within the first 24 hours. Approximately 93.3 percent of the low dose and 99.6 percent of the high dose were eliminated in excreta during the 10-day study period (82.1% and 91.4%, respectively, were eliminated in the urine).

In an additional study, urinary metabolites of alachlor were identified in Rhesus monkeys injected intravenously with 0.7 or 7.0 mg/kg alachlor (Monsanto, 1985a). Five metabolites were identified. They consisted of a secondary and tertiary mercapturate conjugate and a cysteine, thioacetic acid and glucuronide conjugate of alachlor. No side chain hydroxylated metabolites were found. The different doses of alachlor administered appeared to quantitatively but not qualitatively alter the metabolic profile.

Monsanto also conducted two studies (Monsanto, 1985b and c) to examine the interaction of alachlor with blood. The results obtained from these studies are summarized as follows:

- Alachlor is absorbed more rapidly into the circulatory system from oral exposure than it is dermally in male, Long-Evans rats;
- Several metabolites of alachlor bind to the beta-subunit of rat hemoglobin possibly through a reaction with cysteine on the subunit;
- A greater amount of ^{14}C-alachlor equivalents is bound to rat hemoglobin as compared to mouse, monkey and human; and
- Alachlor forms a glutathione conjugate in the red blood cells of mouse, monkey and human.

In the PD-1, the Agency estimated that, based on a 1981 Monsanto dermal absorption Rhesus monkey study, approximately 50% of dermally applied alachlor was absorbed within 24 hours. Additionally, the Agency estimated that dermal absorption of the microencapsulated product could be approximately 12%. In response to the PD-1, Monsanto conducted an additional dermal absorption study in Rhesus monkeys (Monsanto, 1985). This study concluded that dermal absorption is approximately 9.2% and 24.8% for males and 16.0% and 21.8% for females exposed to a tank mix of alachlor. Dermal absorption was estimated to be 4.7% and 8.9% for males and 10.7% and 11.4% for females for concentrated alachlor. Based on this additional data, the Agency's maximum dermal absorption estimate has been revised downwards from the 50% dermal absorption stated in the PD-1 to 24% dermal absorption.

4. Nononcogenic Toxicological Effects

As stated in the PD-1, alachlor exhibits relatively low acute toxicity by the oral (rat LD_{50} = 0.93 g/kg), dermal (rabbit LD_{50} = 13.3 g/kg), or inhalation (rabbit LC_{50} > 5.1 ml/l) routes of exposure (Monsanto, 1978a and 1981c). Although technical alachlor is a skin sensitizer and causes ocular lesions upon chronic exposure, the technical product has only slight skin and eye irritation potential after an acute exposure (Monsanto, 1978b and 1984a).

A 2-year rat feeding study in the Long-Evans strain showed alachlor to be toxic at all doses tested (14.0, 42.0, and 126.0 mg/kg/day) (Monsanto, 1982). The principal nononcogenic toxic effects observed were hepatotoxicity and an ocular lesion, referred to as the uveal degeneration syndrome (UDS). UDS is characterized in its mildest form by free floating iridial and choroidal pigment in the ocular chamber and pigment deposition on the cornea and lens. In its most severe form, the syndrome is characterized by bilateral degeneration of the iris and diminution of the size of the ocular globe with secondary total cataract formation.

A followup 2-year feeding study in the same strain of rat was conducted at 0.5, 2.5, and 15.0 mg/kg/day (Stout, 1983a). There was a small increase at the high dose of animals exhibiting the initial stage of UDS. The 2.5 mg/kg/day was judged to be the NOEL for UDS.

The Agency subsequently received a study in Long-Evans rats run concurrently with the followup 2-year feeding study utilizing a higher dose of alachlor, 126 mg/kg/day, administered for six months (Stout, 1983b). This study indicated that UDS, once established, is an irreversible condition. UDS has not been observed in the other toxicology studies that have been conducted to date.

In a 6-month dog feeding study, alachlor was tested at 5.0, 25.0, 50.0, and 75.0 mg/kg/day and showed dose related hepatotoxicity at all doses (Ahmed, 1981). Increased absolute and relative liver weights were observed at all dose levels for males and at dose levels of 25 mg/kg/day and above for females. Fatty liver degeneration and biliary hyperplasia occurred in both sexes at dose levels of 25 mg/kg/day and greater.

A 1-year oral (by capsule) study was conducted on alachlor in beagle dogs and submitted after issuance of the PD-1. The doses of alachlor were 0, 1.0, 3.0, and 10 mg/kg/day. The lowest observed effect level for alachlor was 3 mg/kg/day based upon hemosiderosis seen in the kidney of one male dog and the spleen of another male dog. At 10 mg/kg/day the effect was also seen in the liver of three out of six male dogs and correlated with hematologic findings showing red cell destruction and consequent red cell replenishment. The hematologic findings noted at 10 mg/kg/day in male dogs were also seen as a trend in the mid-dose dogs. The NOEL for this study was 1.0 mg/kg/day alachlor (Naylor, 1984).

In the three-generation rat reproduction study, alachlor was tested at 3.0, 10.0, and 30.0 mg/kg/day and showed a reproduction NOEL at 10.0 mg/kg/day and reproduction lowest-observed-effect-level (NOEL) at 30.0 mg/kg/day, with indications of renal toxicity observed in F_2 adult males and F_3 pups (Schroeder, 1981). The renal toxicity consisted of kidney discoloration, chronic nephritis and increased relative and absolute kidney weights.

In a teratology study in the rat (Rodwell, 1980), alachlor was administered by gavage at dose levels of 50, 150, and 400 mg/kg/day. A maternal and fetotoxic NOEL was established at 150 mg/kg/day in this study with no teratogenic potential indicated at the highest dose tested, 400 mg/kg/day.

The rabbit teratology study was submitted but was considered inadequate to assess the teratogenic potential of alachlor. The study had the following flaws or confounding problems: inappropriate vehicle, only minimal evidence of maternal toxicity, a high incidence of preimplantation loss in the high dose, inadequate historical data, lack of individual fetal data for body weights and variations as well as other problems (Schardein, 1983).

5. Mutagenicity Findings

Mutagenicity studies conducted on alachlor and discussed in the PD-1 were:

- A rec-assay in *B. subtilis* strains M45 and H17 which was negative;
- Reverse mutation assay in *E. coli* strain WP2 *hcr* and *S. typhimurium* strains TA 1538, TA 1537, TA 98 and TA 100 were negative;
- *In vivo/in vitro* heptacyte DNA repair assay in rats which was weakly positive; and
- An *in vivo* bone marrow cytogenetics study which was negative but considered unacceptable.

After the PD-1 was issued a point mutation assay was conducted on alachlor in Chinese hamster ovary cells which was acceptable as a negative assay.

Several metabolites of alachlor have been tested for mutagenicity in two or more strains of *Salmonella typhimurium* in the Ames assay. A summary of these results can be found in the Table 3.

Table 3. Results of Ames Salmonella Assays on Alachlor Metabolites

	Strain							
Alachlor Metabolite	TA 100		TA 98		TA 1538		TA 1537	
	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9
N-[(2,6-diethyl)phenyl]-N-methoxymethyl-2-(hydroxy) acetamide	-	-	-	-	-	-	-	-
2',6'-diethyl-N-[methoxymethyl]oxanilic acid, sodium salt	-	-	-	-	-	-	-	-
N-[(2,6-diethyl)phenyl] -N-methoxymethyl-2-amino-2-oxaethanesulfonic acid, sodium salt	-	-	-	-	-	-	-	-
3-[N-(2,6-diethyl)phenyl-N-methoxymethyl-2-amino-2-oxoethane-sulfinyl]-2-hydroxypropanoic acid, sodium salt	-	-	-	-	-	-	-	-
N-[2-ethyl-6-(1-hydroxyethyl)phenyl]-N-methoxymethyl)-2-(methylsulfonyl) acetamide	+	+	-	-	-	-	-	-
N-(methoxymethyl)-2-(methylsulfonyl)-2',6'-diethylacetanilide	-	-	-	-	n.t.[1]	n.t.	n.t.	n.t.
N-(methoxymethyl)-2-(methylsulfinyl)-2',6'-diethylacetanilide	-	-	-	-	n.t.	n.t.	n.t.	n.t.
2-(methylsulfinyl)-2',6'-diethylacetanilide	-	-	-	-	n.t.	n.t.	n.t.	n.t.
N-2-ethyl-6-(1-acetoxyethyl)-phenyl-2-(methylsulfonyl) acetamide	-	+[2]	-	-	n.t.	n.t.	n.t.	n.t.
N-2-ethyl-6-(1-hydroxylethyl)-phenyl-2-(methylsulfonyl) acetamide	+	+	-	-	n.t.	n.t.	n.t.	n.t.
N-(methoxymethyl)-2-(methylthio)-2',6'-diethylacetanilide	-	-	-	-	n.t.	n.t.	n.t.	n.t.

[1] n.t. = not tested

[2] weakly positive in TA 100 with metabolic activation

In summary, two metabolites of alachlor, N-[2-ethyl-6-(1-hydroxylethyl)phenyl]-N(methoxymethyl)-2-(methylsulfonyl) acetamide and N-2-ethyl-6-(1-hydroxylethyl)phenyl-2-(methylsulfonyl) acetamide were found to be positive in strain TA 100 both with and without metabolic activation. A third metabolite, N-2-ethyl-6-(1-acetoxyethyl)-phenyl-2(methylsulfonyl) acetamide, was weakly positive in TA 100 in the presence of metabolic activation.

Urine from alachlor treated rats produced a weak mutagenic response in TA 98 without metabolic activation but with beta-glucuronidase, and in TA 1537 in the presence of both metabolic activation and beta-glucuronidase. In TA 100, the Agency questions whether a positive response with urine from alachlor treated rats was not masked due to histidine and other nutrients present in the control urine. This is especially evident when the assay is run in the presence of both S-9 and beta-glucuronidase (Kier, 1985).

Bile from alachlor treated rats did not induce a mutagenic response toward Salmonella strains TA 98, TA 100, TA 1535, and TA 1537 (Kier, 1985).

6. Oncogenicity

In the PD-1 the Agency concluded that alachlor produced oncogenic effects in laboratory animals. The effects included lung tumors in mice and stomach, thyroid, and nasal turbinate tumors in rats. A summary of the test results are described below. No new oncogenicity data were submitted since the issuance of the PD-1.

Male and female mice of the CD-1 strain were fed technical grade alachlor in the diet for 18 months at dosage rates of 26, 78, and 260 mg/kg/day. There was a statistically significant increase ($p < 0.05$) in lung bronchoalveolar tumors at the highest dose tested (Daly, 1981a). The increase of lung tumors in male mice was not significant. Numerical increases were observed in the incidence of liver and uterine tumors in the high-dose group but these increases were not statistically significant. The technical material in this study was stabilized with epichlorohydrin (ECH) (0.5%) for the first 11 months and with another intentionally added inert for the remainder of the study. This change in material tested came about because the registrant removed epichlorohydrin from technical alachlor, replacing it with a different stabilizer.

Three chronic feeding studies were conducted in the Long-Evans strain of rat with alachlor. In the first study, the technical material was stabilized with epichlorohydrin during the first year of the study (Daly, 1981b) and fed to 50 animals/sex at dose levels of 14, 42, and 126 mg/kg/day. During the second year of this study, alachlor was stabilized with another intentionally added inert. Dose-related responses were observed for nasal turbinate tumors of both sexes for the mid and high doses. Also, statistically significant increases were observed in the incidence of malignant stomach tumors (described by the authors as neoplasms pluripotent in ability to form a mixed carcinoma-sarcoma-type tumor) in the high doses of both sexes ($p < 0.001$). In addition, thyroid follicular tumors (adenomas plus carcinomas) increased in both sexes at the high-dosage level with a significant increase in males ($p < 0.001$). The incidences of the nasal turbinate, stomach and thyroid tumors as well as other tumors, (i.e., liver and brain), considered of potential biological significance are shown in Table 4. Table 4 also presents the histological diagnoses of the various tumors.

In the second year of this study (Stout, 1983a), in which an inert other than epichlorohydrin was utilized as a stabilizer in the test material, three treatment groups of 50 male and 50 female Long-Evans rats received 0.5, 2.5, and 15 mg/kg/day. Data from an additional study (Stout, 1983b) which ran concurrently with the previously discussed study used a fourth treatment group, 126 mg/kg/day, that was exposed to the new technical material (with the new stabilizer in place of epichlorohydrin). The design of this study was different from the previous study (Stout, 1983b) because it used a variety of dosing regimens and had the primary purpose of investigating the nature and reversibility of the ocular lesions (UDS). The animals in this additional dose level were subdivided into three groups. One group was sacrificed and examined after approximately 8 months of treatment, one was treated for 5 to 5-1/2 months and then put on a control diet for the remainder of the 2-year period and the third group was treated for the entire 2-year period. The second group was selected from among the larger group primarily on the basis of appearance of early ocular effects rather than random assignment. The biased selection process inherent in the design of this study limits its usefulness for the quantitative assessment of oncogenic potential. However, the results are useful in the qualitative assessment of the weight-of-the-evidence for the oncogenicity of the new technical product not stabilized with epichlorohydrin. This study indicates that the tumor response observed in the earlier study (Daly, 1981b) cannot be explained by the presence of epichlorohydrin in the test material and suggests that partial lifetime exposure (approximately one-fourth of the lifespan of the animals) can result in a similar tumor incidence as a lifetime exposure.

The nasal epithelial adenoma response was statistically significant in both sexes of both 2-year feeding studies ($p < 0.001$). The incidence of this neoplasm in the second chronic rat study is presented in Table 4. In addition to the tumors presented in Table 4, it should be noted that

Table 4. Tumor Incidence in Rats

Dosage mg/kg/day	(Stout 1983b) #ML-80-224 126				(Stout 1983a) #ML-80-186								(Daly 1981b) #BD-77-421							
					0		0.5		2.5		15		0		14		42		126	
	GI*		GIII*																	
No. of tissues examined and incidence of animals with tumors	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Nasal turbinate tumors	61	25	17	46	44	42	47	42	44	47	45	48	46	49	46	47	41	45	42	48
o Adenomas	42†	11†	10	19	0	0	0	0	0	1	15†	14†	0	0	0	0	10†	4	23†	10†
o Adenocarcinoma	7†	2	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0
Stomach	68	31	20	49	50	50	50	50	50	50	49	48	49	50	50	50	50	50	50	50
o Malignant tumors	3	19†	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0	1	17†	23†
- Mixed carcinoma sarcoma	2	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	11†	17†
- Undifferentiated sarcoma	-	5	-	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-
- Undifferentiated carcinoma	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
- Adenocarcinoma	-	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	1
- Leiomyosarcoma	-	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
- Anaplastic sarcoma	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
- Osteosarcoma	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	4
Thyroid	70	31	20	49	49	49	50	49	49	49	49	47	48	49	50	44	49	46	50	49
o C-cell adenoma	4	3	1	2	5	2	3	2	7	4	6	3	4	2	5	3	2	4	7	3
carcinoma	0	0	0	1	0	0	0	0	1	0	0	0	1	0	1	0	0	1	0	0
o Follicular adenoma	8	4	1	3	2	1	4	1	3	0	4	2	1	0	0	0	1	2	11†	2
carcinoma	10†	0	1	1	1	3	0	1	1	1	2	1	0	0	0	0	0	0	2	2
Brain	70	31	20	49	50	50	50	50	50	50	49	49	50	50	50	50	50	50	50	50
o Neuroepithelioma	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
o Ependymoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2	1
Liver	70	31	20	49	50	50	50	50	50	50	50	49	50	50	50	50	50	50	50	50
o Hepatoma	3	1	0	0	1	0	0	1	0	0	0	0	1	0	0	0	2	1	1	3
o Nodular hyperplasia	0	1	0	1	0	0	0	0	0	0	0	1	0	2	0	1	1	2	4	1
o Carcinoma	2	2	0	1	2	0	1	1	2	0	0	2	0	0	0	0	0	0	0	0

*GI - animals maintained on treated diets for 2-years
*GIII - animals maintained on treated diets for only 5 to 6 months
† - statistical significance

there was a submucosal gland adenocarcinoma observed in a mid-dose male in the second study (Stout, 1983a). For risk assessment purposes, this submucosal gland adenocarcinoma was not combined with the adenomas and adenocarcinomas of the nasal turbinate because it was sufficiently different from other nasal tumors observed to not be considered treatment related.

An increase was also noted in the incidence of thyroid follicular cell adenomas or carcinomas in males in the second study, with 4, 4, 6, and 18 (8%, 8%, 12%, and 25%) of the males in the 0.5, 2.5, 15.0, and 126 mg/kg dose groups respectively having this effect compared to 3 (6%) in the control group. A rare stomach tumor (an undifferentiated sarcoma) was found in a 2.5 mg/kg male and it is considered biologically significant in view of its occurrence in several animals at the 126 mg/kg dose level in the second study. No stomach tumors were observed in control animals in any of these chronic rat studies. Several primary brain tumors were noted in treated animals in both the earlier chronic rat study and in the 126 mg/kg dose level extension of the more recent study. The brain tumors observed in the first study were classified as ependymomas, whereas several neuroepitheliomas were observed in the second study. The increased incidences were not significant compared to the control groups. However, the registrant has concluded that these tumors "were possibly due to, or secondary to, treatment with the compound", apparently due to the rarity of this tumor in Long-Evans rats.

Since the PD-1, Monsanto (1985c) has responded to the Agency's comments on the brain tumors. Monsanto's addendum to the special chronic feeding study in Long-Evans rats contains a re-evaluation of the brain tumors (neuroepitheliomas) found in the study. Three brain tumors were reported, two in female rats and one in males. The re-evaluation indicated, according to the report, that these tumors were extensions of nasal adenocarcinomas and not brain tumors. However, several discrepancies between the original report of this study and the addendum need to be resolved prior to accepting the conclusion reached in the addendum, that alachlor does not induce neuroepitheliomas of the brain.

As stated in the PD-1, epichlorohydrin (ECH), when tested alone, was shown to induce both nasal turbinate tumors and stomach tumors in rats (Laskin, 1980; Konishi, 1980). These ECH-associated nasal turbinate tumors appear to be of similar histogenic origin as the ones noted in the alachlor rat studies. The ECH-associated stomach tumors were not malignant, in contrast to those observed in the studies with alachlor. It is difficult to estimate the contribution, if any, ECH made to the results observed in the rat and mouse studies due to the following:

- ° The rats were of different strains in the alachlor and ECH studies.

- ECH was administered through different routes of exposure (i.e., inhalation exposure (Laskin, 1980); oral exposure through treated water (Konishi, 1980); and oral exposure through treated feed (Daly, 1981b).

- ECH was administered to the animals for different durations.

However, the occurrence of these same tumor types in the second rat study in which a different stabilizer replaced ECH (and its 126 mg/kg/day dose extension), suggests an alachlor-related effect, independent of ECH.

7. Human Studies

The Agency is unaware of any human studies that have investigated the oncogenicity of alachlor. There is one limited epidemiological study which investigated the ocular status of workers in a plant where alachlor was manufactured (Coleman, 1980), but no attempt was made to address oncogenic effects in this study.

8. Weight-of-the-Evidence

The goal of the Hazard Identification step of the cancer risk assessment is to reach a qualitative judgment on the weight-of-the-evidence that alachlor may be a human carcinogen.

The data in Table 5 show that alachlor has demonstrated statistically and biologically significant oncogenic responses at multiple sites at multiple doses in two sexes of one rodent species and at one site at one dose in the one sex of a second species. Several oncogenic observations were replicated in the rat (see Table 4).

Table 5. Summary of Statistically Significant Oncogenic Responses in Alachlor Feeding Studies in Mice and Rats

Mouse	Rat
Lung--at one dose (F)	Nasal turbinates--at three doses (MF)[1] Stomach--at one dose (MF)[1] Thyroid--at one dose (M) Thyroid--at one dose (F)

[1] Seen in more than one study.

All observed stomach tumors were malignant as were several of the tumors observed at other sites. In addition, the incidence of nasal turbinate tumors observed after 2 years in rats exposed for 5 to 6 months demonstrates that less than lifetime exposure is sufficient to elicit an oncogenic response in rats (Stout, 1983b).

As further supporting evidence, it can be noted that alachlor is structurally similar to another substituted acetanilide (metolachlor) which has demonstrated oncogenic effects (neoplastic nodules of the liver (NAS, 1980)) in the Sprague-Dawley strain of rat. Mutagenicity tests of alachlor have produced negative and weakly positive results. In addition, five metabolites of alachlor have produced negative results toward Salmonella typhimurium strains TA 98 and TA 100 (Kier, 1985). Another metabolite was mutagenic toward TA 100 both in the presence and absence of metabolic activation (Kier, 1985). Another alachlor metabolite was weakly mutagenic toward TA 100 in the presence of metabolic activation. Neither of these former metabolites were mutagenic toward TA 98 (Kier, 1985).

After consideration and review of the data and comments submitted in response to the PD-1, the Agency's earlier conclusion that alachlor induces oncogenic effects in rats and mice is reaffirmed. It is also concluded that alachlor should remain classified as a B_2 oncogen, or a "probable human carcinogen."

B. Dose-Response Assessment

The upper bound estimates of excess cancer risk presented in this document are based on extrapolation from tumor rates in both males and females in the rat oncogenicity study without the high dose (i.e., using doses 0, 0.5, 2.5, and 15 mg/kg (Stout, 1983a)). The reasons for this choice and the effect of including other available data sets are discussed below. Extrapolation is done using the linearized, multistage model (computed by the Global 83 computer program). This is in keeping with Agency policy for those chemicals for which information regarding the mechanism of oncogenicity is unknown. Also, in keeping with Agency policy as presented in the Agency's Guidelines for Carcinogen Risk Assessment, the interspecies extrapolation for oncogenic effects is accounted for by expressing the dose in terms of weight of alachlor per body surface area per day, rather than weight per body weight per day (EPA, 1986). The effect of treating interspecies differences this way is to increase the experimental rat estimate of risk approximately fivefold when extrapolating from rats to humans.

The Agency has determined that the data from the rat studies is more appropriately used for dose-response assessment than the information from the mouse study because significant oncogenic responses were observed in both sexes, at multiple dose levels and sites, in multiple experiments in rats, whereas tumors were observed in only one sex, at one dose and site, in one experiment in mice. The Agency "Guidelines for Carcinogen Risk Assessment" recommend that ". . . in the absence of evidence to the contrary, the biologically acceptable data set showing the greatest sensitivity should generally be given the greatest emphasis." In addition, analysis of the one mouse study is complicated by the presense of ECH in the test diet for part of the study; rat data are available that are not subject to this limitation.

Because tumors in rats were observed at several sites in both sexes of two separate studies, there were a large number of possible approaches to a quantitative risk assessment. The result of using various data sets and combinations are shown in Table 6. The Q_1^* is a parameter of the linearized multistage extrapolation model. It is used as a multiplier of the estimated exposure (in units of mg/kg/day) to obtain the estimated 95 percent upper bound on risk. A change in the Q_1^* will result in a proportional change in risk.

The entries of Table 6 show that the choice of the data set from among those available for rat oncogenicity makes little difference in the risk estimate. With one exception, the range of the Q_1^*'s shown in that table is less than an order of magnitude. The greatest Q_1^* (1×10^{-1}) is generated using either the nasal turbinate tumor in the female rat (second study without high dose) or by using combined nasal turbinate, thyroid, and stomach tumors from the second study with or without the high dose for each sex or both sexes combined.

The first alachlor rat study provided results qualitatively similar to those produced in the second study. The two studies differed quantitatively, however, as discussed in Chapter II.A.6. The second study is considered the more appropriate for risk assessment because it was conducted with the current technical alachlor mixture and because it was designed and conducted with knowledge that nasal tumors were likely. The Agency considered combining the results of these two studies and the effect of doing so is shown in Table 6. The range of upper bound risk estimates would vary less than twofold from the range given in the risk tables presented in this document. Since the upper bound risks are presented as order of magnitude ranges, this degree of difference makes no meaningful difference in the risk ranges discussed in this document.

Table 6. Q_1* Potency Estimates for Alachlor Based on Rat Tumor Data

NASAL TURBINATES					TURBINATES, STOMACH OR MALE THYROID (FOLLICULAR Ad/Ca)			
		Combining Sexes					Combining Sexes	
Males	Females	Program Input	Geometric Mean	Study Data	Males	Females	Program Input	Geometric Mean
4×10^{-2}	1×10^{-2}	2×10^{-2}	2×10^{-2}	First Study	3×10^{-3}	2×10^{-2}	2×10^{-2}	2×10^{-2}
9×10^{-2}	7×10^{-2}	8×10^{-2}	8×10^{-2}	Second Study	1×10^{-1}	1×10^{-1}	1×10^{-1}	1×10^{-1}
5×10^{-2}	1×10^{-1}	6×10^{-2}	8×10^{-2}	Second Study without High Dose	1×10^{-1}	1×10^{-1}	9×10^{-2}	1×10^{-1}
6×10^{-2}	3×10^{-2}	4×10^{-2}	4×10^{-2}	Combined Studies	6×10^{-2}	4×10^{-2}	5×10^{-2}	5×10^{-2}
5×10^{-2}	2×10^{-2}	3×10^{-2}	4×10^{-2}	Combined without Second Study High-Dose	5×10^{-2}	4×10^{-2}	4×10^{-2}	5×10^{-2}

As previously noted, the additional dose level (126 mg/kg) (Stout, 1983b) was used in the second chronic rat study for the purpose of investigating the reversibility of eye lesions previously observed in the earlier rat study. Because of the criterion employed in assigning animals to the various subgroups in this study (discussed in Chapter II.A.6. of this document), this dose level is not considered fully appropriate for quantitative risk assessment. However, if it were included, the effect would be a slight increase in the Q_1* for males and a decrease for females (less than twofold), leaving a range similar to, but somewhat less than, that actually used by the Agency. As discussed above, this variation is well within the range of uncertainty generally associated with a quantitative risk assessment.

Table 6 also illustrates the effect of combining or separating the tumor incidences observed in males and females.

The combination of data for sexes may be done in either of two ways. First, when data sets for both sexes are essentially equivalent, the data sets are entered together into the computer program and the combined result yields a flattened slope as a result of the increased sample size. Second, when there is some meaningful difference in the dose response patterns displayed by the sexes, the calculation of the geometric mean of two or more Q_1*'s is appropriate. Table 6 presents potency estimates using both of these approaches for each data set (program input and geometric mean, respectively).

Table 6 thus illustrates 40 different approaches to the risk extrapolation from the chronic rat studies to man. It can be readily seen from Table 6 that the use of other subsets of the data yield potency estimates which are similar to that derived from the use of the second chronic study excluding the 126 mg/kg dose level. For example, potency estimates derived from both studies and all dose levels combined are 6×10^{-2} (males) and 3×10^{-2} (females). In fact, all potency estimates are within a fairly narrow range.

The risk tables which follow in this document present the range of upper bound estimates of excess cancer risk generated by extrapolation from tumor rates for both males and females in the second rat oncogenicity study without the high dose (i.e., using doses of 0, 0.5, 2.5, and 15 mg/kg). The upper end of the range represents extrapolation from nasal turbinate tumors in females as well as extrapolation from the combined tumor totals (nasal turbinate, thyroid and stomach) of either sex, (i.e., these approaches yield identical potency estimates). The lower end of the range represents extrapolation from the incidence of nasal turbinate tumors observed in males.

C. Exposure Assessment

1. Dietary Exposures

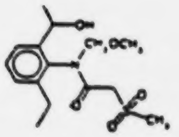
a. Residues from Treated Raw Agricultural Commodities

Alachlor is registered primarily for pre-emergence use on beans (dry), corn, cotton, lima beans, peanuts, peas, sorghum, soybeans, sunflowers, and woody ornamentals. In conjunction with these registrations, tolerances for alachlor residues have been established for the food crop and animal feed commodities associated with these uses (see page 4). Tolerances for alachlor have been established for eggs, milk, and fat, meat, and meat byproducts of cattle, goats, hogs, horses, poultry, sheep and for the food crops listed above. The residue levels permitted under the alachlor tolerances include residues of alachlor, *per se*, plus its metabolites.

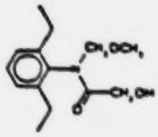
The metabolism of alachlor in plants and livestock was discussed in the PD-1. Additional livestock metabolism data and analytical methodology have been received by the Agency subsequent to the issuance of the PD-1.

As discussed in the PD-1, alachlor is absorbed and translocated by plants. Two groups of alachlor plant metabolites have been identified: those containing the DEA moiety and those containing the HEEA moiety. Structures of these alachlor metabolites are shown in Figure 3. Analytical methods have been developed by Monsanto to determine both types of alachlor metabolites.

Figure 3.



hydroxyethyl-methyl
sulfone metabolite
(contains HEEA moiety)



2-hydroxy analog
of alachlor
(contains DEA moiety)

Since the publication of the PD-1, additional data on the metabolism of alachlor in domestic animals have been received by the Agency. The metabolism of alachlor, however, is still not adequately understood. Available data demonstrate that both the DEA and HEEA metabolites of alachlor are present in meat, milk, poultry, and eggs. However, residues were only partially characterized in milk, eggs and liver, and were not characterized in other tissues. Monsanto has been asked to further characterize the residues in goat and poultry liver, eggs and possibly milk.

Additional residue data have been received from Monsanto subsequent to the issuance of the PD-1. Monsanto submitted residue data for corn and soybeans, processing studies on soybeans, and animal feeding studies. These studies were generated using analytical methods with lower limits of detection than those of the analytical methods previously used. Additionally, both DEA and HEEA metabolites of alachlor were analyzed. The data generated from the new studies enabled the Agency to revise the best available estimates (BAE) for these commodities. Specifically, for corn, soybean oil, meat, milk, poultry, and eggs, the BAE's were lowered. For soybean grain and defatted flour, the BAE's were increased based on the additional data from the two groups of metabolites, in spite of a reduction in the application rate from 8 lb ai/A to 4 lb ai/A. Additionally, residue data from the analysis of both DEA and HEEA metabolites of alachlor were submitted for peanuts. These data showed a higher residue of alachlor and its metabolites which resulted in an increase in the BAE.

No additional residue data were submitted for peas, lima beans, dry beans, cottonseed, sorghum, and sunflower seed. For legume crops, peas, lima beans and dry beans, HEEA metabolite residues were estimated based on residue data from related legume crops, i.e., soybeans and peanuts. The BAE's for these crops were determined to be higher. HEEA metabolite residues for cottonseed, sorghum and sunflower seeds could not be estimated. As such, their BAE's have not changed.

A range of BAE's has been calculated. The upper bound BAE is the maximum expected residue of alachlor and its metabolites resulting from one application of alachlor at the maximum registered rate. The lower bound BAE has been adjusted for percent crop treated (upper bound estimate multiplied by the percent crop treated for crops). The lower bound BAE is used to calculate exposure for the general population. The upper bound BAE is used to represent a worst case estimate of dietary exposure. A worst case estimate of dietary exposure could occur, for example, when a grower whose crops are all treated with alachlor and whose livestock are fed with these treated crops consumes only plant and animal products from his or her own farm. Revised upper and lower bound of residues resulting from registered uses of alachlor are presented in Table 7.

Note that Monsanto data have been used to obtain all of the BAE's. These data, while they are being used for the Special Review, are inadequately validated. Raw data sheets and chromatograms needed to validate the methods, as well

Table 7. Best Available Estimates (BAE) of Alachlor Residues

Crop	Upper Bound BAE (ppm)	% Crop Treated[1]	Lower Bound BAE (ppm)[2]
Corn[3]	0.016	24	0.0038
Soybean grain[3]	0.21	26	0.0546
oil[4]	0.04	26	0.0104
defatted meal[5]	0.27	26	0.0702
defatted flour	0.56[6]	26	0.0130
Peanuts[3]	0.10	56	0.0560
Peas[7]	0.40	3	0.0120
Beans (lima)[7]	0.32	12	0.0384
Beans (dry)[7]	0.36	10	0.0360
Cottonseed[8]	0.02	<1	0.0002
Sorghum[8]	0.05	8	0.0040
Sunflower seed[8]	0.25	2	0.0050
Milk/dairy[3]	0.3 ppb		0.07 ppb[9]
Eggs[3]	0.4 ppb		0.09 ppb[9]
Beef Muscle[3]	0.3 ppb		0.05 ppb[9]
Beef Fat[3]	0.4 ppb		0.09 ppb[9]
Beef Liver[3]	1.7 ppb		0.4 ppb[9]
Beef Kidney[3]	1.8 ppb		0.4 ppb[9]
Poultry Muscle[3]	0.05 ppb		0.01 ppb[9]
Poultry Fat[3]	0.05 ppb		0.01 ppb[9]
Poultry Liver[3]	0.1 ppb		0.03 ppb[9]
Poultry Kidney[3]	0.1 ppb		0.03 ppb[9]
Pork Muscle[3]	0.1 ppb		0.02 ppb[9]
Pork Fat[3]	0.3 ppb		0.03 ppb[9]
Pork Liver[3]	0.4 ppb		0.05 ppb[9]
Pork Kidney[3]	0.4 ppb		0.05 ppb[9]

[1] These percentages were presented in the November 5, 1985 memorandum of R. Petrie and R. Torla to M. Loftus.

[2] The lower bound BAE is obtained by multiplying the upper bound BAE by the percent crop treated.

[3] The BAE is based on residue data where both the DEA and HEEA metabolites were determined for sweet corn.

[4] Estimate for refined, deodorized oil.

[5] Obtained by fractionation of dehulled soybean grain into defatted soybean meal and crude oil.

[6] Average of value obtained from Monsanto residue data for soybean protein concentrates and isolates.

[7] Only the DEA metabolites have been determined. The upper bound BAE is calculated by multiplying the DEA residue level by four (4). Thus, the BAE reflects both DEA and HEEA metabolites.

[8] The BAE reflects residues from DEA metabolites only.

[9] The lower bound BAE represents livestock fed diets reflecting the percent crop treated.

as storage stability data for most crops have not yet been submitted. Additionally, data for HEEA metabolites of alachlor are not available for all crops and the metabolism of alachlor in livestock has not been adequately delineated.

A brief discussion of BAE's and alachlor usage for each crop follows.

Corn, Soybeans and Peanuts

- For corn, soybeans, and peanuts, residue data on both the DEA and HEEA metabolites were available and the residue levels presented in Table 7 for these crops reflect residues from both groups of metabolites.

- The upper and lower bound BAE's for corn, peanuts and soybeans reflects only one treatment at 4 lbs ai/A.

- For peanut, corn and soybean acreage < 1% receive a total dose in excess of 4 lbs ai/A.

- For corn, 3 labels allowed 2 treatments at $\leq$ 8 lbs ai/A, not to exceed a total dose of $\overline{8}$ lbs ai/A.

- For soybeans and peanuts, 3 labels allow 1 to 2 treatments at $\leq$ 4 lbs ai/A .

Lima Beans, Dry Beans and Peas

- For lima beans, dry beans, and peas, data were available only for the DEA metabolites of alachlor. Data for the HEEA metabolites were unavailable for these crops.

- Residues reflecting both DEA and HEEA metabolites in/on lima beans, dry beans, and peas were estimated based on data for related legume crops (soybeans and peanuts). This was done as follows: Since in soybeans and peanuts, HEEA metabolites comprised 75 percent of the total DEA + HEEA metabolites, the maximum residue of DEA metabolites found in lima beans, dry beans, and peas was multiplied by a factor of four to arrive at the upper bound BAE for lima beans, dry beans, and peas.

- Only one treatment at $\leq$ 4 lbs ai/A is registered.

Cottonseed, Sorghum and Sunflower Seed

- For cottonseed, sorghum and sunflower seed, only the DEA metabolites were analyzed and residues of HEEA metabolites were not estimated.

- The BAEs presented in Table 7 for cottonseed, sorghum, and sunflower seed reflect residues of DEA metabolites only. Thus, exposure to alachlor residues from these crops may be underestimated. These data were not corrected to include residues of HEEA metabolites in addition to the DEA metabolites because, either the actual residue of DEA metabolites was unknown (i.e., was reported as nondetectable), or because residue and/or metabolism data for both DEA and HEEA metabolite residues in related crops were unavailable. Thus the ratio of these two types of metabolites could not be calculated.

- Only one treatment at $\leq$ 4 lb ai/A is registered.

Meat, Milk, Poultry and Eggs

- For meat, milk, poultry, and eggs, residue data were available on both DEA and HEEA metabolites. BAE's were calculated, assuming that feeding of peanut forage and hay was restricted. This restriction was assumed because Monsanto has agreed not to seek a tolerance for peanut forage and hay.

- The lower bound BAE represents the average residue expected assuming that livestock were fed feed reflecting the percent crop treated in the United States.

  The upper bound BAE represents the average residue expected assuming that livestock are always fed with treated feed (worst case). Additionally, since the metabolism of alachlor in animals is not adequately understood, these residue estimates are only tentative.

b. Exposure Through Drinking Water

(1) Ground Water

(a) Monitoring

In the PD-1, EPA stated the concern about ground water contamination based on monitoring data from three states (Iowa, Maryland, and Nebraska) and samples from Ontario, Canada, and the potential for alachlor to leach through the soil.

EPA has obtained additional ground water monitoring results since the PD-1 was issued. Most of the additional data have been generated by state agencies. The new data, together with the original ground water monitoring results, are summarized in Table 8A and 8B, respectively. Four observations can be made.

- The true extent of alachlor ground water contamination is not known.
- A significant number of public wells may be contaminated with alachlor residues.
- Improper use, disposal etc., seems to have occurred in many of the cases where alachlor was detected.
- Concentrations associated with normal alachlor use rarely exceed 10 ppb, and are typically between 0.2 and 2.0 ppb.

Each of these observations is discussed below.

- In order to properly estimate the true extent of the occurrence of alachlor residues in well-water, a survey could be done where a representative sample of wells is chosen based on proper statistical, hydrogeological, and agronomic considerations. Monsanto is currently conducting a study in accordance with a protocol approved by EPA. The primary goal of this study is to estimate the true extent of well-water contamination in alachlor use counties as well

Table 8A. Update of Data on Alachlor in Wells

Location	Positive Wells / Total Wells	Concentration (ppb): mean; range	Well Information	Comments
Massachusetts (Massachusetts, 1986)	5/147	1.3; 0.2 to 4.4	Private wells; towns of Westport, Whately and Worthington.	Sampling in potato area only; no sampling in prime use area - corn
Ontario, Canada (Ontario, 1985)	21/305 ("positives"-> 1.0 ppb)	9; 1 to >100	All private wells, many dug wells.	22 additional wells between 0.5 and 1.0 ppb; 12 or 13 wells appeared to be contaminated by surface runoff or spills (spills implicated in 2-4 of these).
Minnesota	1/27	0.66; 0.42 to 0.91 (2 samples 1 month apart)	Public well, 316' deep, 52' deep casing 150' depth to water; limestone aquifer.	Goodhue County; moderately permeable soils; 500-600 people served; most likely due to normal agricultural use.
North Carolina	1/1	6	Dug well, 30' deep	Northhampton County; next to treated fields; also contained heavy metals & benzene; likely due to surface contamination or spills.
Pennsylvania (Buchanan, et al. 1984)	6/82	0.45; 0.1 to 1.8	Private and/or observation-wells; limestone & dolomite; depth to water generally 30' to 60'.	Lancaster and Berks Counties; most likely due to normal agricultural use; moderately use; permeable soils.

Table 8A. Update of Data on Alachlor in Wells (Cont'd)

Location	Positive Wells/Total Wells	Concentration (ppb): mean; range	Well Information	Comments
Iowa (Kelley & Wnuk, 1986; Kelley, 1986)	7/72 (5 systems)	0.2; 0.12 to 0.30 (normal use) 18; 2.5 to 70.0 (spills implicated)	Public wells; alluvial	Western & north-central Iowa; spills/disposal implicated in 3 wells (2 systems); moderately permeable soils; 23,500 people served by the public systems where normal use is implicated.
Iowa (Hallberg, 1986; Hallberg, 1985)	12/?**	105; 1.6 to 580	Private, public, and industrial wells; aquifer types include sand and gravel as well as limestone.	Various locations around the state; almost all positives.
Kansas (1985)	0/58	(MDL=0.4ppb)*	Both irrigation and smaller domestic wells were sampled.	
Nebraska (1985)	0/75	(MDL=1.0ppb)*	All large, deep, irrigation wells. Unconsolidated gravels, silts, and sands of the High Plains Aquifer.	Atrazine and Alachlor used heavily on corn in areas sampled. There were positive findings of Atrazine (18/75 wells sampled).
Illinois (Felsot, 1983)	19/25	0.16; 0.02 to 0.33	Domestic wells. Mostly sandy soils.	Kankakee, Mason, Whiteside, and Gallatin Counties (sandy soil) and Stephenson County (bedrock outcropping). Most likely due to normal agricultural use.

* MDL = minimum detection limit. These two values, especially the 1.0 ppb MDL, are higher than usual, and are in the range of what is usually found.

** These 12 positive wells were found in 7 "sites". It is unknown how many total well were sampled in this program (personal communication, Hallberg, (1986).

Table 8B. Summary of Data on Alachlor in Wells from the PD 1

Location	Positive Samples / Total	Conc. (ppb): mean positives, range	Comments
Ontario, Canada (Frank, 1984)	10/2	2.1; 0.01 to 9.1	Rural well water 19790-1984 tests; all positive wells were shallow.
Ontario, Canada (Frank, 1984)	8/153	147; 0.6 to 780.0	Rural well water 1979-1984 tests; 7 of 8 were result of spills
Nebraska (Spalding et al. 1980)	2/14	.04; .018 to .071	Central Platte Region; 1 to 2 m silt loam above sand and gravel; nitrate found.
Devonian-Carbonate Aquifer, Floyd and Mitchell Co. Iowa (Libra et al. 1984)	8/93	8.3; 0.10 to 16.6	Major findings in Incipient Karst well; water level is 3 to 5 m below surface.
Big Spring Basin, Clayton County, Iowa (Hallberg et al. 1983)	3/35	.09; 0.05 to 0.15	Groundwater discharge into Big Spring.
	1/59	.05	Study area wells.
	4/5	5.1; 0.3 to 12.7	Surface water draining into sinkholes.
Maryland (Maryland DHMH, 1983)	4/30	0.4; 0.1 to 0.8	Wells located in agricultural areas reflecting statewide distribution.

as in various subgroups of wells in those counties (e.g., wells in high-use and high-vulnerability areas). The secondary goal, which would only occur if there were a relatively large number of findings of alachlor in wells, is to correlate the findings with observation and design variables such as well-screen depths, proximity to alachlor use, county-level vulnerability scores, etc.

- Based on the available data, the relatively large number of public wells shown to be affected (15 out of 140), compared to the number of private and observation wells (78) is of concern to the Agency. Even if wells where spills are implicated are removed from consideration, the proportion is still relatively high. For other pesticides, instances of public well contamination have been relatively rare compared with what is found in private wells. Public wells are usually deeper than private wells, usually draw from water deeper in a given aquifer than private wells, are usually better constructed and reduce the chances of surficial contamination down the borehole. These wells, by definition, serve more people than private wells. Thus, findings of alachlor in public wells usually have the following connotations: there is significant alachlor penetration to depths below the surface (relative to occurrence at the water table alone), the occurrence most likely did not occur via seepage down the well boreholes from the surface due to the construction of the wells, and potentially large numbers of people could be exposed. For these reasons EPA is especially concerned about the occurrence of alachlor in public wells.

- Much of the well contamination observed to date may be attributed to spills or holding ponds in association with farm chemical dealers, blending operations, etc. Alachlor is usually used as a liquid formulation and is frequently blended with fertilizers and atrazine. (In fact, many of the water samples containing alachlor also had atrazine). As noted in the PD-1, alachlor is sufficiently mobile and persistent to leach to ground water. It is also a very large volume chemical - 80 and 84 million pounds per year are used in the U.S. It logically follows that whenever large volumes of a chemical are used and mixed in liquid formulations with other substances that some degree of spillage is to be expected. If the chemical is also a leacher, then random occurrences of residues in wells are also expected. In the case of alachlor, where residues in the low parts per billion range in drinking water could be cause for concern, these "occurrences" could often be expected at toxicologically significant concentrations. For purposes of this discussion, an "occurrence" is any confirmed detection.

- Alachlor concentrations in public wells have only exceeded 1 ppb where improper use and/or disposal is suspected. In private well occurrences, this level is frequently exceeded even where normal agricultural use is the likely source. Findings of alachlor in public wells are generally less than 1 ppb when normal use is undertaken.

(b) Mathematical Modeling

Since the publication of the PD-1, the appropriate use of models to determine the leaching potential of alachlor has been reevaluated.

Mathematical models can be used to predict pesticide transport through the unsaturated zone of soil in order to determine the potential of a pesticide to eventually contaminate ground water. One such model is the Pesticide Root Zone Model (PRZM) which was developed at the U.S. EPA Environmental Research Laboratory (Carsel, et al., 1985; Carsel, et al., 1984). It is a one-dimensional, time-varying model for evaluating pesticide fate and transport within the root zone and some specified distance below the root zone within the unsaturated zone. Efforts to validate the PRZM model with actual field data are now beginning to appear in the literature (Carsel, et al., 1985; Lorber and Offutt, 1986; Jones, 1986). Additionally, a 5-year EPA study was initiated in 1983 to study pesticide movement through soil under actual field conditions and to provide a comprehensive data base for testing the PRZM model. However, there has not been a field study on alachlor which would provide a data base for validating the PRZM model on alachlor.

In the absence of such data, parameter values for alachlor were determined from available sources, including the open literature and environmental fate studies provided by Monsanto. The estimates developed can be considered "reasonable worst case" in that they are realistic for alachlor and represent hydrogeologically vulnerable circumstances. Specifically, applications of 3.0 lb ai/A, less than the maximum registered rate, of alachlor were made to corn in Iowa, and soybeans in Indiana and Georgia. The applications were assumed to be pre-plant incorporated and the soil type modeled can be described as a "sand". While these estimates do not represent the bulk of alachlor usage, they do represent vulnerable situations which occur to some extent.

The results of this exercise are given in Table 9. As can be seen, less than 0.01% of applied alachlor was predicted to leach below the corn root zone, but 1.5% and 4.6% was predicted to leach below the soybean root zone in Georgia and Indiana, respectively. The major difference between soybean and corn simulations was that corn root zones were assumed to be 90 cm deep, whereas soybean root zones were assumed to be 45 cm deep (Carsel, et al., 1984). Microbial decay, which is the major mode of alachlor degradation, was assumed to be active to the entire depth of the root zone. Below the root zone, a much slower rate of degradation was assumed. Therefore, alachlor was simulated to have degraded to negligible amounts before reaching 90 cm in the corn simulations, whereas significant amounts leached below 45 cm in the soybean simulations. The major difference between Georgia and Indiana simulations was that the rate of degradation of alachlor in the soybean root zone was assumed to be more rapid in Georgia than in Indiana. This was based on the assumption that microbial degradation would be more rapid in the warmer climate of Georgia as compared to Indiana. For all three scenarios, less than

Table 9. Summary of PRZM Results for Alachlor Applied to Corn in Iowa and Soybeans in Indiana and Georgia

Description	Corn Iowa	Soybeans Indiana	 Georgia
1. Percent of applied alachlor leaching below root zone, %	<0.01	4.6	1.5
2. Percent of applied alachlor leaching below 2 meters, %	<0.01	<0.01	<0.01
3. Average peak soil water concentration of alachlor at 2 meters, ppb.	<0.5	<0.5	<0.5

0.01% of applied alachlor was simulated to leach below two meters in the soil, and soluble concentrations at two meters were consistently less than 1 ppb.

A conclusion that can be drawn from these simulations is that alachlor has the potential to leach below soybean root zones, but in all likelihood would be retained within the deeper corn root zones until microbial degradation resulted in complete dissipation. The ultimate fate of residues below the root zone is a function of mobility and degradation characteristics of residues, in combination with soil characteristics. The assumptions made for these simulations resulted in degradation of residues before two meters. Other assumptions may result in different conclusions. For example, in a sensitivity analysis simulation with PRZM in which the mobility characteristics of alachlor were increased, the amount leaving the Indiana soybean root zone increased from 4.6 to 23.2% and soluble concentrations reached 4.4 ppb at the 2 meter depth in the soil. PRZM also can be limiting in its treatment of water flow. It assumes a horizontally uniform soil profile, and cannot simulate the occurrence of leaching through sinkholes, fractured clay layers, wormholes, or other preferential flow paths which can lead to direct transmission of residues to ground water.

In summary, it is reasonable to conclude that alachlor has demonstrated a potential to leach through the soil and contaminate drinking water sources. As seen in Table 8A and 8B, alachlor has been found in wells, and the causes of these findings have ranged from accidental spills to seepage through sinkholes to infiltration through soil from normal agricultural use. Further, model simulations with the PRZM confirm that alachlor has the potential to leach below soybean root zones, although probably not below the deeper root zones characteristic of corn root zones. This conclusion is reinforced by the monitoring data.

(2) Surface Water

The PD-1 concluded that the estimated lifetime exposure to alachlor from contaminated surface water was between 2.0 to 5.0 ppb for the cornbelt area, based upon a significant amount of monitoring data in northwestern Ohio and limited data in other states and based upon a mathematical modeling assessment.

The PD-1 included several tables of monitoring data. Additional monitoring data have been incorporated into Tables A through E, which are included in the Appendix. Table A contains a summary of surface water monitoring data extracted from a number of reports. Table B contains a summary of alachlor monitoring data obtained from the STORET data base, a publicly-accessible data base maintained by the Agency (EPA, 1982). Alachlor concentrations in drinking water (tap water) and rainwater are shown in Tables C and D, respectively.

The mean concentrations presented in Tables A, C, and D were calculated as simple arithmetic means of all samples and do not represent average daily or seasonal exposures. The value of 0.00 was assigned to samples where alachlor concentration was less than the limit of detection of the analytical method. The limits of detection of the analytical method for alachlor in water is typically 0.15 to 0.25 ppb, although lower limits have been obtained in individual studies.

Much of the data in Tables A through D were developed by David Baker of Heidelberg College in Tiffin, Ohio, who has monitored concentrations of pesticides in northwestern Ohio rivers for several years. Dr. Baker's sampling scheme is stratified such that samples are collected at frequent intervals during periods of expected high concentrations and less often during periods of expected low concentrations. In Table A, the mean concentration values, in which each sample is given equal weight, include a statistical bias toward the samples collected at short intervals--in this case, samples with high concentrations.

In order to remove this bias, a time-weighted average can be calculated for a river or water supply. A time-weighted average, which takes into account varying sampling frequencies, is calculated as follows:

$$\text{Time-weighted average} = (C_{twa}) = \frac{c_i t_i}{t_i}$$

where c_i is the concentration in i^{th} sample and t_i is the duration of time that the i^{th} sample characterizes the system.

The Agency agrees with Dr. Baker's contention that time-weighted averages represent more accurately the actual seasonal or annual exposures to alachlor during the sampling period than the unweighted averages or mean concentrations shown in Table 10 of PD-1. However, the ability for time-weighted averages to characterize actual exposures is enhanced by the number of samples collected. Some sites have too few samples for even a time-weighted average to have meaning.

Dr. Baker calculated time-weighted averages by two different methods. One method allows a sample value to characterize the system for one-half of the time back to the previous sample and one-half of the way forward to the succeeding sample, with no time limit imposed on when the previous and succeeding samples were taken. The other method allows the sample value to characterize the system in a similar fashion back to the previous sample and forward to the succeeding sample, but with a maximum time limit of one day. Both cover the entire year's data, but the number of days that comprise the average are different. The total monitoring time, even with no time factor applied, is the number of days in the sampling period, which is usually less than 365 days. Dr. Baker stated that his data support the assumption of zero concentrations of alachlor during non-monitored periods, since alachlor was not detected at either the beginning or ending sample of the monitoring season.

Dr. Baker's two calculated time-weighted averages for the Maumee and Sandusky Rivers and Honey Creek for the years 1983, 1984, and 1985 in comparison with mean concentrations are shown here (as extracted from Table A):

	Mean Conc. ppb			TWA no time limit ppb			TWA 1-day limit ppb		
	1983	1984	1985	1983	1984	1985	1983	1984	1985
Maumee River	1.60	2.53	0.43	0.45	0.74	0.20	1.47	1.95	0.49
Sandusky River	0.78	1.54	2.85	0.25	0.53	0.78	0.76	1.50	2.44
Honey Creek	1.41	2.02	3.00	0.66	0.88	0.91	1.60	2.01	2.13

As indicated in the PD-1, surface water monitoring studies in several rivers in northwestern Ohio show the presence of alachlor at all sampling stations during the summer months of 1981 to 1985 with peak concentrations occurring consistently between late May and early June. The areas where these monitoring studies were carried out are dominated by corn and soybean production. In general, the highest pesticide concentrations were found in the rivers during the first major runoff events following spring planting. However, the findings of alachlor residues in surface waters two months and longer after application is evidence that alachlor persists in streams or persists in fields for subsequent runoff.

In 1983, simultaneous monitoring in Ohio of the Maumee and Sandusky Rivers and tap water from three municipal water supplies deriving their water from these rivers showed average alachlor levels of 3.11 and 1.24 ppb in the rivers and 1.87, 0.22, and 1.08 ppb in tap water at Bowling Green, Fremont, and Tiffin, respectively, during the period from May 28 to July 27. Powdered activated carbon was used at the Bowling Green treatment Plant from June 1-14, 1983, and achieved 43% removal during that period. A granular activated carbon filter at the Fremont plant removed a significant amount of alachlor in 1983, in contrast to the Tiffin plant (Baker, 1984a). In 1984, the peak concentrations reported were 18.5 ppb on May 24 on the Maumee River compared to 12.25 ppb on May 25 at Bowling Green, and 9.10 ppb on June 28 on the Sandusky River compared to 10.51 ppb at Fremont and 13.65 ppb at Tiffin, both on June 27 (Baker, 1986b). The data indicate that alachlor is not removed by conventional water treatment processes which do not include carbon filtration.

Less than 1 percent of the wells, both public and private, which supply drinking water in the United States are equipped with carbon filtration. Few of the municipal water supply systems in the United States are equipped with carbon filters, although some use powdered activated carbon for removal of disagreeable taste and odors.

As reported in PD-1, the peak concentrations in surface water (e.g., 267.7 ppb at Four-Mile Creek (Iowa) Watershed Site #4 and 104.6 ppb at Defiance, Ohio) were accountable to runoff events. The site in Iowa was located on a creek in close proximity to agricultural fields and was part of a 5-year study to provide information on field-to-stream transport of pesticides for testing and evaluating mathematical models. The site was selected to determine what ambient concentrations could occur in nearfield locations. In two consecutive years, high percentages of positive values were reported at this site (205/222 and 101/104), and the mean concentration in one of those years was 20.3 ppb. This site does not, however, represent a source of drinking water (Johnson, 1982). The concentration of alachlor in runoff depends upon the intensity and magnitude of rainfall and the amount of alachlor present in the field during a rainy day.

Since the publication of the Registration Standard and the PD-1, Monsanto conducted a surface water monitoring program in 1985 to determine actual alachlor levels in samples of raw and finished water from 24 community water systems (CWS) located in regions of high alachlor use in seven States (Iowa, Illinois, Indiana, Montana, Michigan, North Carolina, and Ohio). The concentrations of alachlor were determined in weekly composites of daily water samples with a screening method based on capillary gas chromatography with electron capture detection (GC-ECD). This was followed by a confirmation method of gas chromatography-mass spectrometry (GC-MS) for positive samples. The limit of detection of the screening methods was 0.20 ug/l. Samples were collected for the entire calendar year (1985), which justified calculation of the annualized mean concentration (AMC) of alachlor in each location.

Table E in Appendix A shows the range of AMC's. Monsanto calculated the low end of the range of AMC's by assigning a value of 0 mg/l to weekly samples with non-detectable residues. The high end of the range was calculated by assigning the value of 0.2 mg/l, which is equal to the limit of detection, to weekly samples with non-detectable residues. Briefly, the Monsanto study indicates that:

- Positive findings of alachlor occurred in raw and finished water in 1985 in 14 of the 24 CWS.

- The highest weekly concentration of alachlor was 10.9 ppb and the highest AMC was 1.3 to 1.5 ppb, both of which occurred at Columbus, Ohio.

- The maximum weekly concentration occurred during May or June, following the peak use season of alachlor, and declined rapidly.

- Alachlor was found above 2 ppb in 2.6 percent of all weekly composite samples.

- No correlation was found between the amount of alachlor found and the use pattern.

- No significant difference in the amount of alachlor was found between corresponding raw and finished water in all water treatment plants sampled.

° The geographical distribution of alachlor concentrations is as follows:

  - high use areas in Indiana and Ohio had higher AMC's of alachlor than apparently similar areas in Iowa and Illinois;

  - alachlor was not detected in the CWS (Michigan City, Monroe, and Toledo) using water from the Great Lakes; and

  - very low AMC's of 0.01 ppb to 0.23 ppb were found in CWS (Davenport, Lexington, Mount Vernon, and Quincy) using water from major midwestern rivers.

Monsanto is conducting further monitoring in 1986 which is designed to determine whether soil type, as well as alachlor use, influence where concentrations of alachlor occur in surface water.

The results of an intensive modeling assessment (Mulkey and Donigian, 1984) of alachlor in surface water in Iowa, Mississippi, and Georgia were included in the PD-1. Several useful conclusions were drawn in the PD-1 from the modeling effort. These conclusions are unchanged after consideration of the rebuttal comments, although the Agency acknowledges that some of the assumptions in the modeling were based on Agency judgment which may differ from the judgment of others. The PD-1 concluded that estimates of 2 to 5 ppb appear realistic for lifetime exposure based on alachlor contamination of surface water sources for the cornbelt area based on the Iowa River (Iowa) assessment. Lifetime exposure in the range of 12 ppb is probably more realistic under normal patterns. Based on the Yazoo (MS) and Little River (GA) assessment, the PD-1 concluded that estimates should generally not exceed 1 ppb for lifetime exposure outside the cornbelt area.

As stated in the PD-1, for risk assessment purposes, the modeling estimates of alachlor concentrations greatly increase the confidence that the Agency can place in its estimates of drinking water exposure derived from the available monitoring data on alachlor contamination of surface water sources and of tap water taken from those sources. The closeness between modeled and observed results, the opportunity to consider the effects of important variables such as rainfall or extent of use, the geographic and climatic variation among modeled sites, and careful model and data development all contribute to the usefulness of the results.

In summary, it can be stated that concentrations of alachlor have been detected in surface water runoff in Ohio, Kansas, Iowa and Ontario, Canada. The peak concentrations generally appear during the early summer to midsummer period when alachlor is used in agricultural practice.

As predicted by mathematical modeling and confirmed by monitoring data, estimates of 1 to 2 ppb for long-term exposure to alachlor from contamination of drinking water appear realistic in some areas of the cornbelt region. Current information is insufficient to determine the conditions, other than high alachlor use, which will cause the highest long-term exposure to alachlor through drinking water.

2. Applicator Exposure

In the PD-1, applicator exposure was determined by using Monsanto patch data. The patch data measured worker exposure to alachlor during aerial and ground applications of the emulsifiable concentrate, and microencapsulated and granular formulations. Dermal exposure was assessed by measuring residue deposits on gloves or gauze pads; and respiratory exposure was assessed through the extraction of foam plugs, silica gel and charcoal from air samples taken near the breathing zone. The exposure estimates assumed 50% dermal absorption for the EC and 12% dermal absorption for the microencapsulated and granular formulations with 80 percent protection, 100 percent protection, and no protection from various protective clothing scenarios. The lifetime average exposure to commercial ground applicators from 40 years of exposure for 30 days/year was estimated to be 10^{-3} mg/kg/year. This example assumed 80% protection by clothing.

Subsequent to the issuance of the PD-1, the Agency searched the published literature to obtain surrogate patch data for mixer/loaders handling large agricultural equipment, and for ground boom applicators, pilots and flaggers during aerial applications. Monsanto also submitted biomonitoring data for applicators to supplement the patch data, along with a supporting a monkey metabolism study, and a dermal absorption study.

The monkey metabolism study and the dermal absorption studies are reviewed on pages II-3 to II-4 of this document. The monkey metabolism study confirmed that urinary excretion is the primary route of elimination with 82.1% to 91.4% of alachlor dosage eliminated in the urine. This study also defined the time course for urinary metabolites in monkeys. This information is important because an understanding of pharmacokinetics and metabolism is essential in interpreting biomonitoring data.

New exposure estimates have been determined from Monsanto's biomonitoring data. The Agency believes that biomonitoring data, if supported be adequate and appropriate metabolism studies, generally provide a better measure of actual dosage received in the body than patch data. Passive dosimetry studies use patches of various construction to trap residues which could result in dermal exposure. In contrast, biological monitoring studies estimate the total internal dosage from either a measurement of body burden in selected tissues or fluids, or from the amount of pesticide or degradate excreted from the body. Although both methods have distinct advantages and disadvantages, the major advantage of biological monitoring is that, when the pharmacokinetics and metabolism of a pesticide are understood sufficiently, the actual absorbed dose may be calculated. This major advantage is the rationale for the Agency basing some of its the non-dietary exposure risk estimates on the dosages estimated from the Monsanto biological monitoring study (Reinert, 1986).

a. Surrogate and Monsanto Patch Data

(1) Private Farmer

In the PD-1, the Agency used the following parameters in defining exposure to private ground boom applicators to Lasso EC or Lasso ME using surrogate patch data.

- The average acreage is 190 to 200 acres for corn and 145 to 155 acres for soybeans.
- The majority of private individual farmers will use the 2.5 gallon container of alachlor. Most private farmers will open pour alachlor from those containers and do their own mixing/loading and application. The average application rate of alachlor is 2 to 3 lbs ai/acre at 20 gal/acre.
- The average work day will be 10 hours in length and will include 5 hours a day of actual application time to 120 acres. This is assuming that the farmer has a 400 gallon spray tank that treats 20 acres and requires 50 minutes of actual spray time.
- A private farmer will apply alachlor 1 to 3 days annually. The 3 days is based on the farmer having 355 acres in corn and soybeans.

The exposure estimates based on the published patch data and the Monsanto patch data indicate that during mixing/loading the farmer is estimated to receive a dermal exposure of 4.5 to 13 mg/kg/yr. The farmer will most likely receive a dermal exposure of 1.2 to 3.7 mg/kg/yr during application. This is considered the typical, most likely exposure. Those individuals that use state of the art equipment and are more careful could receive an exposure during application as low as 0.032 to 0.096 mg/kg/yr. Conversely, some individuals could receive application exposures of 31 to 94 mg/kg/yr. The average farmer is expected to do his own mixing/loading and application. Therefore, it is estimated that the farmer will receive a combined exposure that will typically be 5.7 to 17 mg/kg/yr for 1 to 3 days of application annually. The combined exposure can range from a low of 4.5 to 13 mg/kg/yr to a high of 36 to 110 mg/kg/yr.

(2) Commercial Applicators

- Commercial applicators of alachlor will be expected to treat up to 120 acres/day by ground boom application using tractors equipped with 400 gallon tanks. Unlike the private farmer, commercial operations are likely to use a closed loading system with 55 gallon or bulk alachlor containers.
- Application of alachlor to 120 acres will require 5 hours of actual spray time.
- The application rate averages 2 to 3 lbs ai/acre.
- The commercial operator is expected to apply alachlor 15 days per year on the average. This is based on a 3 to 4 week planting season, bad weather one third of the time, and alachlor not being the only herbicide applied during the planting season.

The use of a closed loading system has reduced annual mixer/loader exposure to 1.9 mg/kg/yr. Application exposure is estimated to range from 0.48 to 470 mg/kg/yr with a mean of 19 mg/kg/yr. Again, this range results from variation in application equipment, personal habits, and other factors such as the weather. Those commercial applicators that perform both mixing/loading and application functions are estimated to receive combined exposures of 2.4 to 470 mg/kg/yr with a mean exposure of 21 mg/kg/yr.

(3) Aerial Application

- The average application rate for aerial application is 2.0 to 3.0 lb ai/acre. One pilot can normally treat 80 to 100 acres/hour and spray for 5.5 hours daily. One pilot can treat 480 acres with eight loads. The application crew normally consists of one or two mixer/loaders with the possibility that one mixer/loader can handle more than one plane.
- The average size of a spray tank is 300 gallons and one tank load will treat 60 acres at 5 gal/acre. One mixer/loader is expected to load 4 to 16 tanks/day with the average being 8 loads.
- Aerial application is expected to occur 15 days per year.

The mixer/loader is estimated to receive an average exposure of 7.4 mg/kg/yr using a closed loading system. It is estimated that this exposure can range from 3.7 to 15.0 mg/kg/yr. Pilots are estimated to receive an exposure of 2.4 mg/kg/yr and flaggers an exposure of 23.0 mg/kg/yr.

Aerial application was voluntary removed from the label by Monsanto prior to the issuance of the PD-1. Monsanto's data, submitted in response to the PD-1, supports the conclusion that aerial applicators receive less exposure than ground boom applicators.

(4) Exposure Comparisons between Microencapsulated Formulations and Liquid Formulations

The Agency does not have a data base of published studies in which microencapsulated formulations are used. Mixer/loader and applicator exposure from Monsanto's microencapsulated formulation were compared to the exposure from the liquid formulation in both the Monsanto patch and biomonitoring studies.

When mixer/loaders open poured alachlor, the dermal exposure was 0.077 mg/lb ai. The exposure for open pour Lasso ME was 0.12 mg/lb ai. These exposures are considered essentially the same. Ground boom applicator exposure for both the liquid and ME formulations were nearly identical. Applicator exposure for Lasso was 0.15 mg/hr and for Lasso ME was 0.19

mg/hr. Based on these comparisons, Lasso exposure and Lasso ME exposure during similar application routines should not differ appreciably.

(b) Biomonitoring Exposure Data

Monsanto submitted biomonitoring data. Four Monsanto employees were studied by Monsanto for the Lasso and Lasso Micro-Tech formulations.

- Alachlor was incorporated into corn fields at 4 lbs ai/acre.
- The subjects wore goggles and elbow length rubber gloves during mixing and loading and leather boots, trousers, long sleeve shirt, and cap during the entire operation.
- The mixing/loading involved open pouring from 2.5 gallon containers.
- Enclosed tractor cabs were used during application.

Monitoring of the urine indicated that the estimated internal alachlor dosage for mixing/loading and application of Lasso is 0.0066 ug/kg bwt/lb ai applied. The internal dosage for Lasso Micro-Tech is 0.027 ug/kg bwt/lb ai applied. The dosages represent the highest dosage determined among the four replicates for each formulation. The dosage from either formulation is considered essentially the same.

(c) Conclusions

The Agency pooled the Monsanto patch data with the surrogate patch data compiled from open literature studies (different pesticides but identical application methods) to derive a range of ground boom applicator exposure (0.36 mg/hr to 360.0 mg/hr). Monsanto patch alachlor data is on the low end of the range, 0.36 mg/hr.

The range is three orders of magnitude. The lower end of the range represents workers using the safest techniques and working under calm weather conditions. Conversely, the upper end of the range represents workers using equipment less protective, such as open cab tractors, and who are subject to possible accidents and adverse weather conditions. However, the Agency has noted that in most studies on applicator exposure, the largest variable is personal work habits.

The Agency has also determined that the exposure range for mixer/loaders from the surrogate patch data was approximately an order of magnitude.

The Agency believes that a range of applicator exposure correctly describes real world conditions. In this case, the Agency is assuming a range of two orders of magnitude (0.0066 to 0.66 ug/kg body weight/lb ai) for mixing/loading and application combined based on the biomonitoring data and the Agency's experience with exposure variability. Monsanto's biomonitoring data point, 0.0066 ug/kg/lb ai, is the starting point of this range. The Agency assumes that this is the low end of the range because the biomonitoring study was conducted on Monsanto employees using enclosed cabs. A two order of magnitude range above this point was chosen based on the variation observed in the surrogate patch data. The upper end of this range is a reasonable description of worst case conditions, and the lower end of the range of best case conditions.

The estimated annual internal dosage to individuals mixing/loading and applying alachlor to field crops is based on the biomonitoring study and the previously discussed assumptions. The exposure estimates are as follows:

Private Farmer/1 Day Annually: 2.4×10^{-3} mg/kg/yr to 0.24 mg/kg/yr
Private Farmer/3 Days Annually: 7.0×10^{-3} mg/kg/yr to 0.70 mg/kg/yr
Commercial/15 Days Annually: 0.036 mg/kg/yr to 3.6 mg/kg/yr

D. Risk Characterization

1. Dietary Risks

a. Risks from Consumption of Treated Raw Agricultural Commodities

The dietary risk from consumption of treated raw agricultural commodities is summarized in Table 10. Dietary exposure and risk estimates were calculated using residue data for alachlor and its two major classes of metabolites, DEA and HEEA, for the following crops and food sources: corn, soybeans, meat, milk, poultry, and eggs. For the remaining crops; peas, beans, cottonseed, sorghum, and sunflower seeds, residue data were submitted for alachlor and one class of metabolites (DEA). For peas and dry beans, the expected level of HEEA was added to the calculations based on levels found in related leguminous crops. The risks, utilizing these new data, assuming both 100% crop treated and a best estimate of the actual percent crop treated, are 2×10^{-5} and 2×10^{-6}, respectively. This risk estimate is lower than the dietary exposure risk estimate presented in the PD-1. This is due to the use of new residue data

Table 10. Alachlor Dietary Exposure and Risk Estimates by Crop for United States Population

Commodity	Assuming 100% Crop Treated Exposure[1]	Risk (Upper bound)	Adjusted Exposure[2]	Risk (Upper bound)	Adjusted Exposure[3]	Risk (Upper bound)
Total Exposure (ug/kg/day)	0.169	2×10^{-5}	0.0229	2×10^{-6}	0.0147	1×10^{-6}
	% Exposure/ Risk		% Exposure/ Risk		% Exposure/ Risk	% Crop Treated
Peas	41.0		9.0		4.0	3.0
Beans (Dry)	29.0		32.0		13.0	15.3
Lima Beans	7.0		7.0		3.0	14.0
Peanuts	4.0		17.0		27.0	62.0
Soybeans	9.0		14.0		21.0	21.0
Corn	7.0		18.0		27.0	35.0
Other*	3.6		4.2		2.2	22.0

[1] Exposure assumes 100% of crop is treated. Residues of HEEA metabolites for dry beans, peas, and lima beans are estimated based on HEEA metabolite residues for other leguminous crops.

[2] Exposure is adjusted for % crop treated. The residues of HEEA metabolites for dry beans, peas, and lima beans are estimated based on HEEA metabolite residues for other leguminous crops.

[3] Exposure is adjusted for % crop treated. Residues of HEEA metabolites are not estimated when data are unavailable.

* Meat, milk, poultry, eggs, sorghum, cottonseed, and sunflower seed.

and the consideration of the percent of the crop that is believed to have been treated. The percent of the crop treated represents exposure by the general population as opposed to worst case.

Due to uncertainties in the residue levels present in meat, milk and eggs (which at the time of the PD-1 were believed to be the major contributors to dietary risk), an adjustment for the percentage of crop treated was not conducted in the PD-1. Table 10 presents alachlor dietary exposure and risk three ways: (1) worst case assuming 100% of crop is treated and estimating residue levels of HEEA metabolites for dry beans, peas and lima beans, (2) adjusting residue level for percent crop treated and estimating the amount of HEEA metabolites for dry beans, peas, and lima beans, and (3) adjusting residue levels for percent crop treated without estimating the amount of HEEA metabolites for dry beans, peas, and lima beans. The relative contribution of each crop to the total exposure or risk is also tabulated. These risks were calculated using the Tolerance Assessment System (TAS). For a description of the TAS system refer to the PD-1.

b. Drinking Water Risks

A time-weighted average of 0.1 to 1.0 ppb may be a typical range of exposure for both contaminated surface and ground water. Assuming the consumption of two liters of water per day for a 60-kg adult over the course of a lifetime, the estimated risks from consuming alachlor-contaminated water are shown in Table 11.

Table 11. Assessment of Drinking Water Risks for Alachlor

Exposure Level (ppb)	Upper Limit Estimate of Lifetime Cancer Risk
0.1	4×10^{-7}
0.5	2×10^{-6}
1.0	4×10^{-6}
5.0	2×10^{-5}
10.0	4×10^{-5}

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2. Applicator Upper Limit Risk Estimates

Upper limit risk estimates for applicators, assuming 40 years of exposure in a 70 year lifespan are presented in Table 12. These risk estimates include exposure during mixing/loading. The application rate is assumed to be 4 lbs per acre.

Table 12. Applicator Risk Estimates

Alachlor Use Pattern	Days Per Year	Upper 95% Bound on Risk
Private Farmers	1	4×10^{-5} to 4×10^{-7}
Private Farmers	3	1×10^{-4} to 1×10^{-6}
Commercial Applicators	15	6×10^{-4} to 6×10^{-6}

E. Analysis of Rebuttal Comments Concerning Oncogenic Effects

1. Basis of Presumption

The studies in the Alachlor PD-1 and the Registration Standard (Guidance Document) demonstrated that alachlor is oncogenic in laboratory rats and mice. Exposure to alachlor has resulted in statistically and biologically significant oncogenic responses of multiple sites at multiple doses in two sexes of one rodent species (rat) and at one site at one dose in one sex of a second species (mice).

2. Analysis of Specific Rebuttal Comments Concerning Oncogenicity

The following rebuttal comments concerning oncogenicity were submitted in response to the PD 1 on alachlor. The Agency responses to each comment are also provided. The number in parentheses after the title of each comment is an internal number assigned by the Agency to each comment submitted in response to the PD-1 on alachlor. A listing of all comments submitted is presented in Appendix A. Monsanto Chemical Company submitted detailed comments on the oncogenicity of alachlor. These comments along with the Agency response are summarized below.

Rebuttal Comment 1:

The Agency conclusion that alachlor is oncogenic in mice is not supportable according to Monsanto Chemical Company.

Agency Response:

The major basis for Monsanto's argument is the high spontaneous rate of lung tumors in CD-1 mice. They rely primarily on two literature references to support their position: (Homberger, 1975) and (Sher, 1982).

The Homberger data on lung tumors are given for 102 female CD-1 mice at different ages, at an average of 22.6 months for adenomas and 22.4 months for adenocarcinomas and at 18 months. From the data given, the difference between lung tumor incidence at 22 months and 18 months is quite striking. At an age of 22 months, 24 lung adenomas were reported as compared to only 4 adenomas at 18 months and for adenocarcinomas eight versus one, respectively. The alachlor study was 18 months.

The Sher paper gives tumor incidence data for 1240 female CD-1 mice. According to the table in the paper the length of the studies was from 81 to 105 weeks. There were 24 groups of mice. The percentage incidence of adenomas ranged from 0 to 41 percent and of adenocarcinomas 0 to 12 percent. The average incidence was 14 percent for adenomas and 3 percent for adenocarcinomas.

In considering Monsanto's argument, the Agency also had available historical control data on this strain of mouse from the conducting contract laboratory (Biodynamics). Data on the incidence of alveologenic carcinomas and adenomas in female CD-1 mice were available in six studies with each study having two control groups. The average length of these studies was 24 months. A comparison of these data with that obtained in the Monsanto alachlor oncogenicity study in CD-1 mice is found in Table 13.

Table 13. Lung Tumors (Females Only)

Alachlor Study Dose Group	adenomas	carcinomas	combined
control	2/50 (4%)	1/50 (2%)	3/50 (6%)
low	4/50 (8%)	1/50 (2%)	5/50 (10%)
mid	7/50 (14%)	1/50 (2%)	8/50 (16%)
high	10/50 (20%)	1/50 (2%)	11/50 (22%)
Biodynamics Control Data	60/690 (8.6%)	70/690 (10.1%)	130/690 (18.7%)
Range	0 - 8.4%	0 - 18%	2 - 23%

The Agency agrees that the control rate for lung tumors in the alachlor study appears low when compared to the contemporary historical control data from the contract laboratory; however, historical control data were supplied on studies which were conducted for an average of 6 months longer than the alachlor study. One of Monsanto's own references (Sher, 1982) indicates that there is a dramatic increase in lung tumors in female CD-1 mice between 18 and 22 months.

Furthermore, in the Monsanto study there was also an increased incidence of lung ($p < 0.01$) tumors in alachlor treated female mice that died or were killed *in extremis* during the study.

The Agency cannot accept Monsanto's contention that alachlor is not oncogenic to female CD-1 mice. The fact remains that (1) there was a statistically significant increase in the incidence of lung tumors in alachlor treated female mice ($p < 0.05$) along with an increase in the number of lung tumors in female mice killed *in extremis* ($p < 0.01$) and (2) the incidence of lung tumors in the high dose female mice was slightly above the average historical control rate from the conducting laboratory and was barely within the historical control range, despite the differences in the lengths of the studies (alachlor study, 18 months versus an average of 24 months for the contemporary studies).

Rebuttal Comment 2:

There is substantial evidence that alachlor is not genotoxic or mutagenic.

Agency Response:

The results of mutagenicity testing conducted on alachlor and found to be acceptable by the Agency are summarized in Table 14.

The available data indicate that alachlor was "weakly genotoxic" in an *in vivo*/*in vitro* hepatocyte DNA repair assay and negative in four other assays.

Two metabolites of alachlor, N-[2-ethyl-6-(1-hydroxyethyl)-phenyl]-N-(methoxymethyl)-2-(methylsulfonyl) acetamide, and N-2-ethyl-6-(1-hydroxyethyl)-phenyl-2-(methylsulfonyl) acetamide were tested in the Ames/*Salmonella* assay and found to be positive in strain TA 100 both with and without activation. A third metabolite, N-2-ethyl-6-(lacetoxyethyl)-phenyl-2-(methylsulfonyl)acetamide, was weakly positive in TA 100 in the presence of metabolic activation.

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Table 14. Summary of Alachlor Mutagenicity Studies

Test	Result	Comments
Ames assay	negative	a positive response was seen at 5000 ug/plate in TA 1535 but the response was not repeated for consecutive doses.
Gene mutation in CHO cells at HGPRT locus	negative	
In vivo bone marrow chromosome	negative	no structural or numerical chromosomal aberrations
In vivo - *in vitro* hepatocyte DNA repair assay	positive	positive at highest dose tested (1.0 g/kg), "weakly genotoxic"
DNA damage in *B. subtilis* M45 and H17	negative	did not cause DNA damage (20-20,000 ug/plate)

Rebuttal Comment 3:

Numerous studies demonstrate that alachlor does not produce mutagenic responses, thereby providing supporting evidence that alachlor is not a human carcinogen.

Agency Response:

The Agency cannot concur with this statement and cites the *Report of the NTP Ad Hoc Panel on Chemical Carcinogenesis Testing and Evaluation*, August 17, 1984 in support of their opinion:

". . . because of the limitations of the present battery (for example, relative inability to detect non-initiating/ non-mutagenic carcinogens, the fact that it is not known which endpoint(s) are critical in carcinogenesis, etc.), a lack of positive results, by itself, could not justify a conclusion that a positive animal test was a "false positive." Therefore, for purposes of interpreting animal data, negative short term test data do not by themselves provide a basis for discounting positive results from animal bioassays. As IARC has already done for purposes of classifying a chemical regarding carcinogenicity where animal data were limited, short term test positives can be regarded as support for a conclusion of potential human carcinogenicity based on experimental data. However, a positive bioassay cannot be invalidated or discounted by negative short term data alone."

The EPA's own report (Chemical Carcinogens; A Review of the Science and its Associated Principles, February 1985. FR Vol. 50 No. 50 p. 10372) states, "Short-term tests are presently limited in their ability to predict the presence or absence of carcinogenicity and cannot supplant data from long-term animal studies or epidemiological investigations, since the tests do not necessarily screen for all potential means of cancer induction and do not necessarily mimic all reactions that would occur *in vivo*." This position is consistent with a has been reaffirmed in the Agency's carcinogen risk assessment guidelines (EPA, 1986).

Rebuttal Comment 4:

The results of the rat test do not suggest human oncogenic effects because the rat is not an appropriate model for man in the case of alachlor. Monsanto's argument to support their conclusion is summarized below:

a. The rat is not an appropriate metabolic model for man in the case of alachlor. The monkey would be a better model.

b. The MTD was exceeded at the dosage level of alachlor that induced stomach and thyroid tumors, leaving only nasal turbinate tumors induced at lower dosage levels of alachlor.

c. The other tumor type possibly induced by alachlor at the highest dose tested was neuroepithelioma in brain. Evidence now suggests that these tumors were actually extensions of nasal turbinate adenocarcinomas.

d. Alachlor induces tumors only in one strain of rats. The induction of nasal turbinate tumors in Long-Evans rats is species specific and, therefore, has no relevance to man.

e. A study of the mortality records of workers who manufacture alachlor indicates that there was no association between cancer deaths and the manufacture of alachlor.

Agency Response:

On point a. the following differences in the metabolism of the alachlor in the Sprague-Dawley rat and the monkey can be noted from studies submitted by Monsanto:

1. number of the identifiable urinary metabolites: five in the monkey versus 14 in rat;

2. ratio of urinary to fecal radioactivity recovered: rat 1:1, monkey 9 to 10:1. Monsanto claims that this difference is due to the different molecular weight thresholds for biliary excretion in rats and monkeys (325 ± 50 versus 475 ± 50, respectively);

3. only two metabolites were common to both rat and monkey urine (two mercapturate conjugates);

4. sulfate conjugation and side chain hydroxylation were not metabolic pathways identified for alachlor in the monkey (urine only); and

5. the half-life of elimination in the rat is 8.2 to 10.6 hours for the first phase and 5 to 16 days for the second phase, while the half-life in the monkey was calculated to be approximately 5 hours (considering both the first and second phases of elimination).

In blood binding studies with alachlor, submitted by Monsanto in their rebuttal the following was reported:

1. a greater amount of ^{14}C-alachlor equivalents (alachlor + metabolites) are bound to rat hemoglobin as compared to mouse, monkey, and human; and

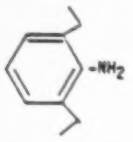
2. alachlor preferentially forms a glutathione conjugate in the red blood cells of the mouse, monkey and human but binds to the beta-subunit of hemoglobin possibly through a cysteine linkage in the rat.

While the Agency would certainly agree that there may be differences in the metabolism of alachlor by rat and monkey, the data that Monsanto submitted to date do not convincingly support the rest of their argument.

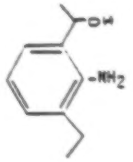
Although the threshold molecular weight for biliary excretion in humans has been estimated to be approximately 500, which is closer to the monkey (475) than the rat (325) (Gillette, 1977), it has not been adequately demonstrated and is only one factor. Examples of cases where compounds are excreted in the bile below that threshold in humans include 15 percent of a given dose of indomethacin (molecular weight = 358), 15 percent of a given dose of diazepam (molecular weight = 285) and 23 to 40 percent of a given dose of practolol (molecular weight = 264) (Rollins, 1984). Thus, molecular weight plays a role in biliary excretion but other factors must also influence it.

Furthermore, while the Rhesus monkey is a good metabolic model for humans for a series of arylacetic acids, e.g., amphetamine and isoniazid; the rat is a better model for humans for oxisurn and 2-acetamidofluorene and is as good as the Rhesus monkey for hydratropic acid and diphenylacetic acid (Smith, 1977). Furthermore, the rates of elimination of drugs are generally lower in humans than in experimental animals including the monkey (Davies, 1977).

In addition, alachlor metabolism in monkeys and humans may be different: the identification of metabolites in the urine of monkeys indicated that only metabolites which contained the DEA moiety were present, while in the human biomonitoring studies, metabolites which contained the HEEA moiety were also present in urine at a level that required attention (i.e., DEA:HEEA was generally 4:1 but in one individual it was 1:2). Hence, the monkey is not an ideal surrogate for humans, and all available data from other animal species should be considered for extrapolation to humans. The DEA and HEEA moieties refer to the following structures:



DEA Moiety



HEEA Moiety

The blood binding studies submitted by Monsanto in their rebuttal are certainly interesting, however, they do not give any insight into the mechanism of cancer induction by alachlor in the Long-Evans rat. As indicated by Monsanto, the toxicological significance of these studies is not known. The studies do, however, raise questions about potential immunotoxicity problems with alachlor.

Monsanto also cites the results of four other studies to support their argument that the monkey is a better model for man than the rat. These include:

1. Alachlor Pharmacokinetics and Metabolism in the Long-Evans Rat;

2. *In vitro* Metabolism of alachlor by Liver and Kidney Homogenates;

3. Alachlor Metabolism in CD-1 Mice; and

4. Animal Whole-Body Autoradiography Studies.

Only summaries of these studies were submitted in the rebuttal and, therefore, it is impossible at this time to adequately evaluate Monsanto's conclusions that were based on the data generated.

Regarding point b. on page II-49, the Agency would agree that the MTD was exceeded at the highest dose (126 mg/kg/day) of alachlor in the Long-Evans rat; however, nasal turbinate tumors were induced at lower levels of alachlor (2.4, 14, and 42 mg/kg/day) and one mixed carcinomasarcoma stomach tumor was induced in a male rat of the 42 mg/kg/day dosage level. The one stomach tumor at this lower level is considered alachlor related because of its unusual etiology.

Monsanto's argument that nasal turbinate tumors induced by alachlor is species-specific since no neoplastic lesions were seen in a one-year beagle dog study (even though nasal and stomach tumors can be induced in the dog) cannot be accepted by the Agency. To make such a conclusion from a dog study of such short duration is not scientifically sound.

Monsanto's argument that nasal turbinate tumors were not induced in the CD-1 mouse is acknowledged. However, it is is quite common for a chemical carcinogen to induce tumors at different sites in different rodent species.

Regarding point c. on page II-49, Monsanto submitted an addendum to the special chronic feeding study (MSL-3492) in Long-Evans rats that contains a reevaluation of the brain tumors (neuroepitheliomas) seen in this study. The only level of alachlor administered in the feed was equivalent to 126 mg/kg/day. Three brain tumors were reported, two in female rats and one in males. Reevaluation, according to the report, indicated that these tumors were extensions of nasal adenocarcinomas and not brain tumors. However, several discrepancies between the original report of this study and the addendum need to be resolved prior to accepting the conclusion reached in the addendum, that alachlor does not induce neuroepitheliomas.

On point d. on page II-49, the Agency cannot agree that alachlor causes tumors in only one strain of rats. Alachlor has been tested for oncogenicity in only one strain of rats and we maintain that alachlor is also oncogenic in female CD-1 mice.

Regarding point e. on page II-49, the limited data discussed by Monsanto on the relation of occupational exposure to alachlor and cancer deaths is not sufficient to conclude that there is "no association between deaths from cancer and the manufacture of alachlor" or that alachlor is not a human carcinogen. Death records on alachlor workers, of the type used by Monsanto to reach their conclusion, are not always reliable for determining the underlying cause of death. Frequently only the immediate cause of death is given. Only a well-designed epidemiology study on alachlor workers is likely to generate the information necessary to make such a decision.

Rebuttal Comment 5:

Monsanto disagrees with the Agency methods for conducting risk assessments. Specifically the comment was "The Agency should utilize risk ranges based on several models and on realistic conditions which reflect a spectrum of exposure situations and not solely on a single model and hypothetical exposure assumptions." (Monsanto rebuttal Volume 2, Appendix A, Part 5).

Agency Response:

The Agency approach to this problem is stated in the current Guidelines for Carcinogen Risk Assessment (EPA, 1986): "An established procedure does not yet exist for making "most likely" or "best" estimates of risk within the range of uncertainty defined by the upper and lower limit estimates. If data and procedures become available, the Agency will also provide "most likely" or "best" estimates of risk. This will be most feasible when human data are available and when exposures are in the dose range of the data."

In the case of alachlor, it is not possible to make a "best estimate" of risk and, therefore, the upper limit of the linearized multistage model is presented.

3. Summary of Rebuttal Comments Concerning Oncogenic Effects

The rebuttal comments received do not rebut the presumption of oncogenicity for alachlor.

F. Analysis of Rebuttal Comments Concerning Exposure

1. Dietary Exposures

a. Residues from Treated Raw Agricultural Commodities

Rebuttal Comment 1:

Dietary exposure was overestimated because residue levels were not corrected for the percentage of crop treated, based on accurate market share data.

Agency Response:

In the PD-1, the Agency did not adjust residue levels for percent crop treated. The Agency has now calculated a range of dietary exposure estimates by using residue levels corrected for percent crop treated to represent the lower bound and residue levels uncorrected for percent crop treated to represent the upper bound dietary exposure estimate. Adjusting for percent crop treated will give an estimate of the dietary exposure for the general population. Assuming that 100 percent of the crop is treated represents a worst case scenario (e.g., a grower whose crops are treated with alachlor, whose livestock are fed with these treated crops, and who consumes only plant and animal products from his or her own farm).

Monsanto and EPA are using slightly different percentages for the percent crop treated. These percentages are compared in Table 15. However, even if the Agency were to use Monsanto's percentage crop figures, it would not substantially affect the Agency's exposure estimates.

Table 15. Comparison of Monsanto and EPA Percent Crop Treated

Crop	Percent	
	Monsanto	EPA
Corn	39	35
Soybeans	29	21
Peanuts	37	62
Peas	10	3
Beans (lima)	10	14
Beans (dry)	10	15.3
Cottonseed	<5	<1
Sorghum	6	8
Sunflower seed	10	2

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Rebuttal Comment 2:

The impact of industrial processing and consumer preparation should be considered in providing a more realistic assessment of dietary exposure.

Agency Response:

Data showing whether there is a reduction of alachlor residues upon processing or consumer preparation were generally unavailable at the time the PD-1 was issued. Monsanto has recently made processing data on soybeans available to EPA. The Agency has reassessed the dietary exposure from soybeans using these data. The effect of processing or consumer preparation on residue levels in beans and peas is unknown. These data have been required from the registrant under the authority of FIFRA section 3(c)(2)(B).

Rebuttal Comment 3:

The use of tolerances for meat, milk, and eggs, set at the limit of detection, to calculate dietary exposure from animal products overestimates the exposure since nondetectable residues were found in animal products. Residue levels in meat, milk, poultry, and eggs used to calculate dietary exposure should reflect the dietary intake of livestock, adjusted for market share (% crop treated). The residue levels should be estimated by extrapolating from the residue levels found in meat, milk, poultry, and eggs for livestock fed at exaggerated dietary intakes down to residue levels reflecting actual livestock intakes.

Agency Response:

At the time the PD-1 was issued, data enabling the Agency to extrapolate meat, milk, poultry, and egg data to lower levels were not available. Monsanto has recently submitted new feeding studies. These recently submitted data were generated using an analytical method with a limit of detection lower than that of the methods previously used. Using these data, the Agency has extrapolated meat, milk, poultry, and egg residue levels from the levels found in feeding livestock at exaggerated rates down to levels reflecting the dietary intake of livestock.

The Agency has calculated a range of exposures. The upper bound calculated by the Agency is based on the average residue expected assuming that livestock always were fed with treated feed (worst case). Additionally, the upper bound is calculated based on a diet of 20 percent soybean forage, 25 percent sorghum forage, 25 percent soybean meal, and 30 percent corn grain for beef cattle; 40 percent soybean forage, 10 percent sorghum forage, 25 percent soybean meal, and 25 percent corn grain for dairy cattle; 50 percent soybean grain, 20 percent soybean meal, 15 percent sunflower meal, and 15 percent corn grain for poultry; and 20 percent soybean meal, 50 percent soybean hulls, 30 percent sorghum forage, and 45 percent sorghum grain for swine. It was assumed that feeding of peanut forage and hay have been restricted.

The upper bound residue estimate for animal products would be expected, for example, for a grower whose feed crops are all treated with alachlor, whose livestock are fed with these treated crops, and who consumes only plant and animal products from his or her own farm. The lower bound of this range is based on the average residue in animal products expected assuming that livestock were fed feed items containing alachlor residues reflecting the percent crop treated in the USA.

The lower bound of dietary exposure calculated by the Agency corresponds to that calculated by Monsanto, except that the Agency and Monsanto accomplish the extrapolation using different methods. The Agency estimated residue levels reflecting actual livestock dietary intake by interpolation. Monsanto estimated residue levels reflecting actual livestock intake by generating least squares dose response curves fitted to a zero y-intercept. (Curves with a y-intercept at zero were observed only for eggs and milk. A nonzero y-intercept was observed for tissues.) At these levels, we do not consider the differences between the Monsanto estimates and the Agency's lower bound estimates to be significant.

In addition, Monsanto calculated dietary exposure for meat using the residue levels for beef, poultry, and pork muscle. The Agency, on the other hand, calculated dietary exposure levels for meat using residue levels for fat, liver, and kidney, in addition to those for muscle (see Table 6).

A comparison of Monsanto and the Agency's residue level estimates for muscle, milk, and eggs is given in Table 16.

Rebuttal Comment 4:

Residue data generated from analytical methods with lower limits of detection should be used in assessing dietary exposure.

Agency Response:

Data from methods with lower limits of detection were submitted to the Agency by Monsanto subsequent to the issuance of the PD-1. These data have been recently submitted and reviewed and have now been considered in the Agency's current estimation of dietary exposure.

Table 16. Comparison of Monsanto and the Agency's Residue Estimates for Meat, Milk, and Eggs

Tissue	Residue (ppb alachlor)	
	Monsanto	EPA
Eggs	0.12	0.09 - 0.4
Milk	0.017	0.07 - 0.3
Beef Muscle	0.0091	0.05 - 0.3
Poultry Muscle	0.0057	0.01 - 0.05
Pork Muscle	0.011	0.02 - 0.1

Note: The nature of the residue is not adequately delineated in livestock. These estimates may underestimate the dietary exposure to alachlor residues in meat, milk, poultry, and eggs.

Rebuttal Comment 5:

Dietary exposure was overestimated because data reflecting application at the 8 lb ai/A rate, instead of the 4 lb ai/A rate were used.

Agency Response:

At the time the PD-1 was issued, the application rate for some crops on registered labels was 8 lb ai/A. Since rates have been reduced to ≤ 4 lb ai/A, or since < 1 percent of the acreage received a yearly dose in excess of 4 lb ai/A, the Agency has recalculated the dietary exposure reflecting rates of ≤ 4 lb ai/A.

Rebuttal Comment 6:

Using the Food Factor system, Monsanto has recalculated dietary exposure using best available estimates of residues for some crops which are different from those presented in the PD-1 or different from those currently used by the Agency.

Agency Response:

Each individual crop is discussed below. Monsanto data were used to obtain what the Agency considers the best available estimates of residue levels. These revised residue estimates are presented in Table 6. Although these data are being used for the purposes of the Special Review, they are inadequately validated. For most crops, raw data sheets and chromatograms needed to validate the methods, as well as storage stability data, have not yet been submitted. In addition, the metabolism of alachlor in livestock has not been adequately delineated (See Section II.C.l.a.).

Corn: In their response to the PD-1, Monsanto indicated that a residue level of 0.01 ppm multiplied by the percent corp treated should be used to estimate the alachlor residue in corn.

In the PD-1, alachlor and its metabolites were estimated at 0.05 ppm (N) for corn. This value reflected 100 percent crop treated and only residues containing the DEA moiety. (The parent, alachlor, in addition to some of its metabolites, contains the DEA moiety.) Subsequent to the issuance of the PD-1, Monsanto submitted residue data for corn, determining both the DEA and HEEA metabolites, and using methodology with lower limits of detection (0.002 ppm LOD for DEA metabolites and 0.002 ppm LOD for HEEA metabolites). On the basis of these new data, the Agency now estimates that residues of alachlor and its DEA and HEEA metabolites in/on corn will generally not exceed 0.016 ppm as the result of registered uses. The value of 0.016 ppm is the maximum residue found in corn following one treatment at 4 lb ai/A. The Agency uses the maximum residue reflecting one treatment at 4 lb ai/A rather than the maximum registered rate reflecting one treatment at 8 lb ai/A or two sequential treatments at 4 lb ai/A because only < 1 percent of the corn acreage receives a total dose in excess of 4 lb ai/A. The residue level of 0.016 ppm for corn reflects 100 percent crop treated and is the best available upper bound estimate for the purposes of calculating the upper bound of dietary exposure. This value is close to the Monsanto estimate of 0.01 ppm.

To obtain the lower bound residue estimate, the Agency has multiplied 0.016 ppm by the percent crop treated of 24 percent.

Soybeans: In their response to the PD-1, Monsanto states that the value for the residue level in soybeans to be used with the Food Factor system should be a weighted average of the residues in soybean oil (93% x 0.005 ppm), meal (4% x 0.09 ppm), and protein concentrates and isolates (3% x 0.005 ppm), multiplied by the percent treated, rather than the residue value for soybean grain assuming 100 percent crop treated.

In the PD-1, alachlor and its metabolites in soybeans were estimated at 0.09 ppm. This value reflected the maximum level expected for soybean grain at 8 lb ai/A, assuming that 100 percent of the crop was treated, and that residues contained the DEA moiety only.

At the time the PD-1 was issued, residue data for processed soybean fractions were not available, nor were data reflecting analysis for HEEA metabolites available. Subsequent to the issuance of the PD-1, Monsanto submitted new residue data where both the DEA and HEEA metabolites were determined, and where soybeans were processed into oil and other fractions. The Agency has used these data to estimate exposure resulting from one treatment of soybeans at the 4 lb ai/A (current maximum use is two treatments at 4 lb ai/A; however, only a negligible percentage of the soybean acreage receives the two treatments). Since these processing data are now available, the Agency agrees with Monsanto that a weighted average of the residues for soybean oil, meal, and protein concentrates and isolates should be used to represent the residue level for soybeans if the Food Factor system is used. In this document, using the newer TAS, the same is accomplished by using the individual residue levels for soybean oil, meal, protein concentrates and protein isolates.

On the basis of data available prior to the issuance of the PD-1 and more recently submitted data, the Agency estimates that residues of alachlor and its DEA and HEEA metabolites will not generally exceed 0.21 ppm in/on soybean grain as a result of registered uses. Monsanto's characterization of the residue in soybeans as "marginally above 0.2 ppm" is certainly consistent with the Agency's recent estimate. Using the value of 0.21 ppm for the alachlor residue in soybean grain and applying average concentration/reduction factors determined from processing studies, the Agency calculates the best available estimate for alachlor and its DEA and HEEA metabolites in human foods: soybean oil, 0.04 ppm, defatted soybean meal (flour and grits), 0.27 ppm, protein concentrates, 0.07 ppm, and protein isolates, 0.04 ppm. These values reflect 100 percent crop treated and are used to calculate an upper bound of dietary exposure. For the lower bound dietary exposure estimate, the Agency has multiplied these values by the percent crop treated, 26 percent.

It is not clear how Monsanto arrived at their residue levels of 0.005 ppm, 0.09 ppm, and 0.005 ppm for soybean oil, meal, and protein concentrates and isolates, respectively, to represent the best available estimates before adjusting for percent crop treated, since both Monsanto and the Agency agree that the best available estimate for soybean grain, before adjusting for percent crop treated is 0.21 ppm.

Peanuts: In their response to the PD-1, Monsanto indicates that a residue level of 0.02 ppm, multiplied by the percent crop treated should be used to estimate dietary exposure from peanuts.

The residue level of 0.02 ppm (N) was used in the PD-1. Nondetectable residues of alachlor and its DEA metabolites had been reported in/on peanuts up to the time the PD-1 was issued. Subsequent to the issuance of the PD-1, Monsanto has submitted additional data for peanuts reflecting analysis for both the DEA and HEEA metabolites. These data show that detectable residues of alachlor and its DEA and HEEA metabolites occur in peanuts, and are not expected to exceed 0.10 ppm following one early treatment at ≤ 4 lb ai/A. Since < 1 percent of the peanut acreage receive more than one treatment or a yearly dose in excess of 4 lb ai/A, the Agency is now using 0.10 ppm as the upper bound best available estimate for peanuts. This value reflects 100 percent crop treated. For the lower bound best available estimate, the Agency has multiplied this value by the percent crop treated, 56 percent.

Peas, Lima Beans, Dry Beans: Monsanto indicates in their response to the PD-1 that the residue levels of 0.10, 0.08, and 0.09 ppm, in peas, lima beans and dry beans, respectively, multiplied by the percent crop treated, should be used to calculate dietary exposure.

The values of 0.10, 0.08, and 0.09 ppm were used in the PD-1 to calculate exposure from peas, lima beans, and dry beans, respectively. Since the issuance of the PD-1, though, the Agency has reevaluated these estimates. The residue levels used in the PD-1 are the maximum residues of alachlor and its DEA metabolites only. These residue levels do not reflect those expected from HEEA metabolites. There are still no available residue data for HEEA metabolites in/on the legume crops peas, lima beans, and dry beans. However, residue data for HEEA metabolites are now available for the legume crops soybeans and peanuts. These residue data for soybeans and peanuts show that only 25 percent of the total residue is from DEA metabolites and that the remaining residue is from HEEA metabolites. On the basis of the residue data for the legume crops soybeans and peanuts, showing a ratio of residue levels of DEA to HEEA metabolites of 1:3, we have multiplied the residue levels of 0.1 ppm for peas, 0.08 for lima beans, and 0.09 ppm for dry beans by a factor of four (4) in order to obtain a best available estimate which would include the HEEA metabolites in addition to the DEA metabolites.

Sorghum: Monsanto indicates that the residue level used to calculate exposure from sorghum should be 0.05 ppm (N) multiplied by the percent crop treated. The level of 0.05 ppm (N) is the level used in the PD-1 and the level currently used by the Agency to represent the upper bound best available estimate. To obtain a lower bound estimate, the Agency has multiplied the upper bound value by percent crop treated. These residue estimates for sorghum reflect DEA metabolites only. Residue data for HEEA metabolites in/on sorghum are still not available. Although residue data for HEEA metabolites in/on the related crop corn are available, and could be applied to sorghum, we have not corrected the 0.05 ppm (N) value for sorghum to include HEEA metabolites; since the actual level of DEA metabolites in/on sorghum is unknown as only nondetectable residues of DEA metabolites have been reported.

Cottonseed: In their response to the PD-1, Monsanto indicates that a value of 0.02 ppm multiplied by the percent crop treated should be used to estimate dietary exposure from cottonseed.

The value of 0.02 ppm (N) for alachlor residues in cottonseed was the value used in the PD-1 as the best available estimate, and is the value currently used by the Agency to calculate the upper bound of dietary exposure. The lower bound estimate is calculated by multiplying this value by percent crop treated. The residue data on which the best available estimate is based do not reflect exposure from HEEA metabolites. Only residue data reflecting analysis for DEA metabolites are available. The Agency is unable to estimate residues of HEEA metabolites in cottonseed because the levels of DEA metabolites are unknown (nondetectable residues of DEA metabolites were found) and because no metabolism or residue data are available for cottonseed or related crops, showing the ratio of DEA and HEEA metabolites.

Sunflower Seed: In their response to PD-1, Monsanto indicates that a value of 0.25 ppm multiplied by percent crop treated should be used to estimate dietary exposure from sunflower seed.

The level of 0.25 ppm was the level used in the PD-1 to estimate dietary exposure from sunflower seed and is the level currently used by the Agency to calculate the upper bound of dietary exposure. This level is the upper bound best available estimate and reflects the maximum expected residue of alachlor and its DEA metabolites resulting from application at the registered rate. It does not reflect exposure from HEEA metabolites. Since no residue or metabolism data are available showing the ratio of DEA and HEEA metabolites in sunflower seed or related crops, the Agency is unable to estimate residues of HEEA metabolites in sunflower seed.

Rebuttal Comment 7:

The metabolism of alachlor in crops and animals is well understood and therefore residues can be fully assessed.

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Agency Response:

EPA agrees that the metabolism in crops is adequately understood. However, the metabolism in ruminants and poultry is not adequately understood because the residue was only partially characterized in milk, eggs, and liver, and was not characterized in other tissues.

Rebuttal Comment 8:

Ciba Geigy responded that Monsanto, in their rebuttal comments calculated the TMRC for both alachlor and metolachlor using different bases for the calculation. Specifically the TMRC for alachlor was calculated ignoring the dietary contribution from potatoes, while the TMRC for metolachlor included the dietary contribution for potatoes. Additionally, the TMRC for alachlor ignored rotational crop tolerances, while the TMRC for metolachlor included rotational crop tolerances, and pending tolerances for metabolites were included. Calculation errors were made as well and the TMRC should be adjusted for percent crop treated.

Agency Response:

The same basis should be used to calculate dietary exposure from alachlor and metolachlor. This document does not include a dietary exposure assessment for metolachlor.

Rebuttal Comment 9:

The metabolism of metolachlor in plants is not adequately defined.

Agency Response:

The metabolism of metolachlor in plants was considered adequately defined and is discussed in the metolachlor Registration Standard. The metabolism of metolachlor may be reassessed in the future.

Rebuttal Comment 10:

EPA states, "Determination of actual health risks from alachlor residues is complicated by lack of adequate analytical methodology . . . the actual levels of residues are uncertain even for the available limited data." It is understandable that the risk from dietary exposure to alachlor on corn is extraordinarily high. One-third of the 2.6 billion bushels of United States corn production, is fed to livestock and poultry on farms where corn is raised. Thus, the highest cancer risks of alachlor are posed by corn, milk and dairy (and meat, poultry, and eggs) and drinking water. The risk from these sources is as high as potatoes, the registration for which was voluntarily withdrawn by Monsanto. The difference is not the risk, but the volume of use. Potatoes were a minor use. Corn constitutes 98 percent of the use of alachlor, i.e., 78 to 82 million pounds and, if withdrawn, would virtually eliminate Monsanto's market.

Agency Response:

The dietary exposure from corn indicated in the Alachlor PD-1 is due to direct exposure. It is not due to indirect exposure by the feeding of corn to livestock. Indirect dietary exposure through corn by the feeding of corn to livestock is accounted for in the tolerance/best available estimate of actual residue in meat, milk, and eggs.

The statement that the highest cancer risk of alachlor, taking into consideration the cancellation of the use on potatoes, is posed by corn, milk and dairy, presumably because the dietary exposure from these products is the highest is correct when dietary exposure is calculated from tolerances, as was done in the PD-1. The Agency has reassessed dietary risk from alachlor use based on data recently submitted by Monsanto. These risks, presented in Table 10, show that corn and meat products do not pose the greatest risks from alachlor dietary exposure, particularly when HEEA metabolites of alachlor are considered.

b. Exposure through Drinking Water from Ground Water Sources

Rebuttal Comment 1:

Monsanto had made an extensive search of the available literature for data which might have been used to validate and calibrate the Pesticide Root Zone Model (PRZM). Monsanto has found no literature reports or data which would substantiate that PRZM has been either validated or calibrated. Monsanto believes that calculations of absolute levels of leaching produced by this model are suspect and produce unrealistic estimates of leachability.

Agency Response:

Publications are in the open literature which document theory and present validation exercises of PRZM (Carsel, 1984; Carsel, 1985; and Lorber and Offutt, 1986).

Furthermore, Union Carbide Corporation has published an evaluation which also utilizes PRZM for aldicarb in a validation and assessment mode (Jones, 1985). Finally, PRZM has been calibrated/ validated within the Agency on other pesticides, where the registrant has submitted field data which can be used in conjunction with PRZM.

Rebuttal Comment 2:

Monsanto concludes that the LEACH methodology, like PRZM, is invalid, and that the parameters used in the LEACH exercise, as well as the environmental conditions assumed are unrealistic.

Agency Response:

Again, since PRZM has been validated in open literature references, and since the LEACH methodology is based on PRZM, it can be concluded that LEACH will produce meaningful results. The LEACH methodology in the PD-1 was removed for the PD-2/3 in deference to the more site-specific PRZM applications.

Rebuttal Comment 3:

Monsanto states that they are "not aware of any studies completed or in progress which will produce data for a calibration of the model for the calculation of absolute amounts of leaching of alachlor in any specific site."

Agency Response:

The Agency is also not aware of any such studies.

Rebuttal Comment 4:

Monsanto states it is known that water can and does move upward through the soil profile. PRZM has no apparent mechanism to account for the effect of moving of water and pesticide upward through the soil column and resubjecting it to other decay mechanisms which would further reduce the amount of material reaching the bottom of the soil profile.

Agency Response:

The Agency agrees with Monsanto's point that PRZM does not account for the upward movement of water through the soil column. However, this phenomena is prevalent in clay soils or soils of similar texture which hold water tightly and in which this "wick" effect occurs. These are not the type of soils in which pesticides commonly leach, nor the type of soils in which PRZM purports to be able to model. Furthermore, the simulations reported in PD-1 are on sand soils, in which this phenomena would occur only in the upper 4 to 6 inches of soil as part of the surface soil evaporation process.

Rebuttal Comment 5:

Monsanto basically disagrees with the choice of parameters used in the PRZM simulations, and repeats the simulations using parameters which they feel more accurately describe the majority of alachlor use sites as well as more accurately describe alachlor itself. Specifically, Monsanto alters the field capacity, the assumption of incorporating the pesticide, the adsorption partition coefficient, and the decay rate of alachlor in their attempt to show that the model will show essentially no leaching when their choice of parameters which are "more representative of the bulk of LASSO usage" are substituted for the Agency choices.

Agency Response:

The following is a justification of the Agency parameters and a response to their substitute parameters. The Agency simulations are an attempt to simulate a "reasonable worst case".

Field capacity: Monsanto substitutes a value of 0.20 cm^3/cm^3 in place of the Agency's choice of 0.09 cm^3/cm^3. Monsanto's suggestion clearly changes the assumption of a sand soil to an assumption of a loam soil, which would hold more water and from a solute transport point of view, would reduce leaching. Monsanto's analysis (p. 126) shows that changing the field capacity from 0.09 to 0.20 while leaving all other parameters unchanged reduces the leaching below a soybean root zone from 4.0 percent to 1.1 percent. Since the objective is to simulate a sand soil, the Agency's choice of 0.09 cm^3/cm^3 is reasonable. That value is not uncommon for agricultural sand soils, with some agricultural sand soils having a field capacity as low as 0.05.

Adsorption partition coefficient: Monsanto argued that the choice of 0.7 for a partition coefficient, K_d, was too low, and that a range of 8.0 to 23.0 is more valid. The results of studies in the Agency's files support the choice of 0.7 as a K_d for sand soils. [K_d values are soil-water distribution coefficients; the lower the K_d value, the more mobile the chemical and the more likely it is to leach].

1) K_d values were determined for alachlor for a Sharpsburg silty clay loam, Jansen sandy loam, and Jansen gravelly sand (pH 6.3, 0.07% organic matter, 93% sand, 2% silt, and 5% clay). Resultant K_d's for the Jansen gravelly sand ranged from 0.70 to 0.94 for the different solution concentrations (Lavy, 1974).

2) Alachlor was leached through columns containing Lakeland sand (99.1% sand, 0.5% silt, 0.4% clay, 0.4% organic matter), Collembey sand, a silt loam, and a sandy clay loam, with 8 inches of water. Alachlor leached to depths of 4 cm, 10 cm, 18 cm, and > 30 cm in the sandy loam, silt loam, Collembey sand, and Lakeland sand, respectively (Guth, 1975).

3) Freundlich K values were determined for a Plainfield sand (89% sand, 6% silt, 5% clay, 0.8% organic matter), a Plano silt loam, and a Sebewa sandy loam. Freundlich K values for the Plainfield sand, the Plano silt loam, and the Sebewa sandy loam soil were 0.5 ($1/n = 0.88$), 1.3 ($1/n = 0.92$), and 7.1 ($1/n - 0.97$), respectively (Harvey, Jordon, 1978).

4) From the same study listed above, additional information that the K_d (in contrast to the Freundlich K) for the Plainfield sand was 0.62. Other soils reported on which had K_d less than 2.0 were: Plano silt loam 1.52, Withee silt loam - 1.97, Fayetee silt loam - 1.47, Peebles clay - 1.63, and Kewaunee clay loam - 1.06 (Jordon, Harvey, 1978).

Finally, justification for a K_d of 0.7 can be made using information Monsanto used to determine appropriate LEACH inputs. They cited five empirical formulae listed in the LEACH manual to determine K_{oc} (organic carbon adsorption coefficient) given K_{ow} (octanol:water partition coefficient). In fact, there are six formulae listed in the LEACH manual for this purpose. Assuming a K_{ow} of 434, they estimate a K_{oc} of 350.3 based on an average K_{oc} determined from the five formulae. Averaging the sixth formula gives a K_{oc} of 337. Assuming an average organic matter (OM) content of 0.5 to 1.0 percent for a sand soil and the relationship that $OC = OM/1.724$, the range of K_d for a sand soil would be 0.98 to 1.95. If only the sixth formula (which leads to the lowest estimate of K_d when compared to the other five) is used in the same way to estimate K_d, than the range of K_d would be 0.50 to 1.00 for a sand soil.

Monsanto presents evidence as to why the K_d is more appropriately 8.3 to 23.8. They do so assuming a more recent estimate of K_{ow} of 1220. More importantly; however, they do so by attempting to estimate "average" organic carbon for Indiana. Their methodology is valid assuming there are no other than "average" soil types in Indiana and elsewhere. If instead this methodology is repeated, assuming K_{ow} is 1220, and instead the range of organic carbon is 0.29 to 0.58 (this assumes organic matter ranges from 0.5 to 1.0), than the resultant range of K_d is 0.86 to 6.59 (this also uses the sixth formula in the leach model not recognized by Monsanto).

In summary, available studies have determined K_d's of less than 1.0 for low organic matter sandy soils. Estimation of K_d for alachlor for sand soils based on k_{ow} and empirical formulae relating K_{ow} to K_{oc} leads to a range of K_d of 0.86 to 6.59. All reference in Monsanto's rebuttal to appropriate ranges of K_d for alachlor of 8.3 to 23.8 assumes "average" soil types, which have higher organic matter content than sandy soils. Given that the PRZM simulations are based on the intention to simulate "reasonable worst case" sandy soils, the choice of K_d of 0.7 is justified.

Decay rate: Apparently, there is not much information in the literature on the decay rate of alachlor. Unlike the many references Monsanto cites on adsorption partition coefficient, they only cite one reference which supports their choice of the decay rate constant of 0.0384 day^{-1} in place of 0.0165 day^{-1}. Their reference was an article entitled, "The Pesticide Content of Surface Water Draining from Agricultural Fields - A Review" by R.D. Wauchope, which appeared in the Journal of Environmental Quality in 1978. In this article is a table listing pesticides found in surface runoff, and estimates of each pesticide's solubility and persistance. Persistance is expressed as 90 percent disappearance on this table, and alachlor's persistance is listed as 2 months, which calculates to a decay rate of 0.0384 day^{-1}. The reference for this estimate by Wauchope is an EPA Document entitled, "Control of Water Pollution From Cropland Volume 1 A Manual for Guideline Development" which also has a table listing alachlor's persistance (90% disappearance) as 40 to 70 days. There is no reference for this value. With this guidance from the literature, Monsanto claims that their choice of decay rate represents a "more accurate and realistic value . . ." than the choice made by the Agency. The choice of 0.0165 day^{-1} was determined from an article by Cohen et al. ("Potential Pesticide Contamination of Groundwater from Agricultural Uses" ACS Symposium Series #259 1984) which lists the alachlor soil half-life as between 1 and 10 weeks; 0.0165 day^{-1} is equivalent to 7 weeks.

Monsanto does not comment on the decay rate assumed for residues which leach below the root zone. In the PRZM simulations, this decay rate constant was set at 0.00346 day^{-1} (half-life = 200 days). Indeed, in Monsanto's suggested replacement tables for PRZM simulations found on p. 91, they only simulate to the bottom of the root zone. This is not surprising since all the evidence indicates that alachlor's primary mode of decay is microbial, and microbial decay decreases with depth, and can be considered low to nonexistent below the root zone.

The following articles support this rate of decay for alachlor residues which leach below the root zone:

1) In anaerobic soil metabolism studies, alachlor was not significantly metabolized to CO_2 from treated soils. After a 12-week incubation period, evolved $^{14}CO_2$

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accounted for 0.1 to 0.3 percent of ^{14}C initially applied to three soils as ring-labeled [^{14}C]-alachlor at 10 ppm; alachlor accounted for $\leq$ 56 percent of the initial ^{14}C (Sutherland et. al., 1972).

2) In this study, [^{14}C] alachlor was added at 0.072 and 10 ppm to ground water samples from four Nebraska locations and incubated in the dark at 10 or 25 °C for 0, 2, 4, 6, 9, 12, or 15 months. Although the results at 0.072 were variable, the samples at 10 ppm showed little loss of ^{14}C between the 0 and 15 month incubation period (Lavy, 1974).

In summary, the choice of 0.0165 day^{-1} as the decay rate in the root zone is justifiable. An examination of the literature indicates that there is not much data to support Monsanto's claim that 0.0384 day^{-1} is a "more realistic and accurate" estimate of decay. Also, the rate of decay of 0.00346 day^{-1} assumed for residues below the root zone is justifiable.

Alachlor incorporation: Monsanto claims that this practice is not common, and that assumption of incorporation to a depth of 10 cm will lead to overestimation of the amount of alachlor leaching. Incorporation to a depth of 10 cm results in applications equally spread between the top 2 to 5-cm zones, rather than evenly spread in the top 5-cm zone, which is the PRZM assumption for surface applications. There is little effect on model predictions of mass leaving a 45 cm root zone with a surface application in comparison to an application incorporated to 10 cm. Monsanto performed simulations on corn and soybeans with all parameters identical to Agency-assumed parameters except this depth of incorporation assumptions. Their results showed 4.0 percent to leave the root zone for soybeans, in contrast to Agency predictions of 4.6 percent, and 0.0098 percent to leave the corn root zone in contrast to < 0.01 percent. Since they used different weather records than the Agency simulations, the difference between 4.0 and 4.6 percent is not definitive, but it can be reasonably concluded that a change in this assumption would lead to a reduction in predictions of mass leaving the root zone by about 10 percent. Most importantly, this would not change any conclusions drawn by the Agency in the PD-1, i.e., that model simulations indicate the potential for leaching by alachlor, with or without incorporation.

Rebuttal Comment 6:

Monsanto correlated model predictions of mass leaving the root zone to ground water concentrations. Monsanto stated, "Leaching of even 0.01% . . .", would seem to mean that "even with the highest prediction of leaching", ground water concentrations are still lower than could be meaningful.

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Agency Response:

If Monsanto had done the calculation with the 4.6 percent leaving the soybean root zone and then concluded that ground water concentrations are low at 0.3 ppb, their statement would be logical. However, if their calculations are duplicated with 4.6 percent assumed for the amount leaching below the root zone, than the predicted ground water concentration would be 138 ppb which is highly significant.

Rebuttal Comment 7:

Monsanto disagrees with the Agency's determination that alachlor is mobile. Monsanto states that the K_d values 0.6 to 8.1 reported in the PD-1 were calculated primarily on the basis of the octanol/water partition coefficient.

Agency Response:

The K_d values in the PD-1 were not based primarily on the octanol/water partition coefficient. The basis of the K_d values, is the 1984 paper by Cohen, et al. as cited in the PD-1. This paper quoted actual K_d values derived from soil studies submitted in support of registrations. No estimates were used, only soil sorption measurements. The Kd values which have been reviewed were based on studies with approximately 12 soils, ranging from silty clay loams to gravelly sand. Therefore, Monsanto's calculated K_d values of 8 to 23 are less appropriate than the laboratory determined range of 0.6 to 8.1. The conclusion that alachlor is mobile in most soils remains.

Rebuttal Comment 8:

Monsanto argues that the PD-1 estimated alachlor contamination of ground water in the Big Spring Basin was due to surface runoff into the spring.

Agency Response:

Monsanto attempts to rebut the Big Spring Basin results by arguing that positive samples are due to direct surface runoff into the spring. The spring is located in and surrounded by woodlands, not treated crops. It is, therefore, highly improbable, if not impossible, for alachlor to be in the surface runoff into the spring. However, the recharge for the spring is derived from precipitation falling on the 103 sq. miles of Karst topography in the Big Spring Basin. Undoubtedly, some of this recharge is from surface runoff which moves through sinkholes and surface depressions. However, the positive samples at the spring discharge are highly unlikely to be from direct surface runoff into the spring discharge structure.

Rebuttal Comment 9:

Monsanto argues that the use of Table 7 in the PD-1 (Tables 8a and 9b in the PD-2/3) is inappropriate because it includes data on wells known to be contaminated from spills and the general misuse of pesticides, including alachlor.

Agency Response:

Table 7 was used to demonstrate that alachlor can move through the soil and leach to ground water. Table 7 points out where spills have occurred, the depth of the wells sampled, and the soil types. Table 7 illustrates that there appear to be two primary sources for alachlor to leach to ground water - normal use and handling problems.

Rebuttal Comment 10:

Monsanto proposes to replace Table 7 in the PD-1 with a new table prepared by Monsanto.

Agency Response:

Regarding Monsanto's replacement table, a general caution on the presentation of negative data is warranted. Negative well data are only significant if it can be established that the chemical was used close enough to the sampled well so that there is a reasonable hydrologic connection between the chemical use-area and the sampling point on the water table. This information is not always apparent in the suggested replacement table.

c. Exposure through Drinking Water from Surface Water Sources

Rebuttal Comment 1:

David Baker of Heidelberg College in Tiffin, Ohio, stated that 66 percent of the surface water data and 75 percent of the drinking water data for alachlor cited in Tables 11 and 13, respectively, in the Alachlor Position Document 1 were from his studies of pesticides transport in northwestern Ohio Rivers. The sampling programs in his studies were biased to collect the water samples more frequently during the expected high concentration periods in order to obtain data on peak pesticide concentrations and pesticide transport. Dr. Baker objects to the calculated average concentration values in the PD-1 and suggests using time-weighted averages to take into account varying sampling frequencies. Dr. Baker summarized the data for three stations for three years assuming zero concentra-

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tions of alachlor during unmonitored periods. Dr. Baker stated that his data support this assumption of zero concentration since alachlor was not detected at either the beginning or ending period of the monitoring season. Dr. Baker contends that the time weighted average will provide more realistic estimates of average daily and seasonal exposure than the mean concentrations calculated by the Agency in the PD-1.

Agency Response:

The Agency was aware of the limited nature of the existing monitoring data at the time of publication of the PD-1. As can be seen on page II-32, the Agency has incorporated Dr. Baker's time-weighted average theory into the overall exposure analysis.

There were few annual pesticide monitoring studies and no statistically designed sampling programs to monitor the concentrations of alachlor in surface, ground, and drinking water corresponding to low, medium, and high use patterns.

In 1985 Monsanto conducted a monitoring program for alachlor, which has added to the existing data base on drinking water supplies using surface water.

Rebuttal Comment 2:

The National Audubon Society stated that even if alachlor were microencapsulated, alachlor would migrate to water regardless of the tillage practice. They further state that the Agency does not need to wait for adequate analytical methodology to estimate exposure.

Agency Response:

Adequate analytical methods are needed to evaluate the effect of microencapsulation on the amount of alachlor available for exposure.

Rebuttal Comment 3:

According to the National Audubon Society, soil incorporation is unlikely to sufficiently reduce the movement of alachlor to surface water. Alachlor is primarily transported by water; and, according to Dr. Baker, even conservation tillage with its surface residue is not effective in reducing runoff losses of alachlor. Increased applications are often necessary as a result.

Agency Response:

Dr. Baker's statement concerning conservation tillage is not supported by data.

Rebuttal Comment 4:

Why is the Agency requesting Monsanto to monitor ground and surface water when the Agency knows alachlor is in ground water supplies?

Agency Response:

Monsanto argues that the amount of alachlor present in drinking water is insignificant or below "the no effect level" on the basis of an annualized calculation from the available seasonal monitoring data for well water and surface water. The Agency has requested that Monsanto supply annual monitoring data corresponding to use patterns of low, medium, and high to address this issue.

Rebuttal Comment 5:

National Audubon Society cites Baker's data in which alachlor has been found in surface water in amounts that are consistent and alarmingly high. The maximum alachlor concentrations are 0.1 ppm in water and 1 ppm in sediment when 28 percent of the watershed was treated with alachlor.

Agency Response:

The maximum level is associated with a particular combination of intensity and duration of rainfall which is not expected to occur more than once or twice per year. The Agency considered the full range of ambient conditions, including Dr. Baker's data, in developing its risk assessment.

Rebuttal Comment 6:

Modeling assessment does not reflect actual fields where there are holes in the soils, root channels, animal burrows etc. Rice and Bowman of USDA/ARS reported that USDA models and predictions have grossly overestimated the rate pesticides leach through soil and ground water. Alachlor travels through sandy soils, silt loam (Nebraska), Karst topographies (Iowa), and big pores into sink holes (Iowa).

Agency Response:

Presently, available models do not simulate worm holes, root channels, or animal burrows. As such, models may underestimate leaching because field conditions, such as those cited above, can lead to the rapid transport of pesticides to the ground water at the onset of a storm. EPA's PRZM simulation showed that alachlor has the potential to leach and supplements the limited available ground water monitoring data.

Rebuttal Comment 7:

Monsanto has submitted rebuttal documents which included studies and reports pertaining to the use of alachlor in conservation tillage versus conventional tillage.

Agency Response:

Additional descriptive side-by-side studies are needed to conclusively show the differences in pesticide runoff from conventional and conservation tillage fields for alachlor and other herbicides used in these agronomic practices.

Rebuttal Comment 8:

According to Monsanto, "The Agency has also used uncalibrated and unvalidated computer programs to model runoff or leaching and thus give more credence to its position."

Agency Response:

The Hydrologic Simulation Program Fortran (HSPF) model is the best available model for studying non-point source runoff from farmlands. The calibration and verification results for the alachlor concentration at Tracer, IA are shown in Donigian and Mulkey (1984). Thus, EPA believes that the HSPF model has been calibrated and validated.

Rebuttal Comment 9:

"As a matter of fact, is should be understood that most alachlor use is correlated with areas of low population. Most people receive water from surface sites far removed from alachlor use or from deep wells located near regions of high use. Little or no alachlor is expected in these drinking water sources. Alachlor has been found in surface and ground water only on a sporadic basis."

Agency Response:

The preliminary results of Monsanto's 1985 monitoring study indicate that urban populations can, in fact, be exposed to alachlor through drinking water sources. For example, Monsanto's monitoring data show the highest concentration detected was 11 ppb, and the range of annualized mean concentration was 1.2 to < 1.3 ppb at Columbus, Ohio (Monsanto, 1986).

Rebuttal Comment 10:

The large majority of samples were collected during the peak alachlor use season. In order to measure peak concentrations, samples collected around rainfall events are analyzed with higher frequency than those collected at other times. This type of sampling and analysis plan cannot be used to show that alachlor is found in nearly every sample analyzed.

Agency Response:

It is true that the available data were collected during the peak alachlor use season. This was either to study trend analyses of pesticide runoff events or to calibrate and verify the output data of mathematical models under development to accurately predict pesticide runoff. Monsanto is conducting a year-round drinking water monitoring project to analyze weekly composite samples for 1986. The concept of yearly monitoring has been agreed to by the Agency.

Rebuttal Comment 11:

Table 10 of PD-1 needs to be replaced because the simple arithmetic mean concentrations reported therein are misleading and do not represent time-weighted average concentrations. The time-weighted mean concentration of alachlor was calculated by assuming zero concentration for unmeasured days.

Agency Response:

The reporting of time-weighted average concentrations instead of arithmetic mean concentrations in Table 10 of PD-1 (Table A in Appendix A of the TSD) would not change the seasonal or specific day alachlor exposure via drinking water. When Monsanto's 1986 monitoring program is completed, the results may allow insight into the proposed annualized time-weighted average concept.

Rebuttal Comment 12:

"This major, multi-year study of 11 watersheds in southern Ontario describes alachlor as 'rarely' seen (< 1% of samples), and when observed its presence can be attributed to 'carelessness, termed spills'" (Frank et al., 1984).

Agency Response:

This statement does accurately reflect the results of the Canadian study. This multi-year survey study (May 1975 - April 1977) of 11 watersheds in Canada resulted in finding alachlor in < 1 percent of the total samples analyzed, and the positive data were from the spring season, and then only in a spill situation. Frank et al. do not report the following: (1) GC-MS confirmation; (2) recovery and reproducibility; (3) limit of detection of gas chromatographic method; (4) water sampling time referenced to spray operation time; and, (5) underestimation for low level alachlor measurements.

Rebuttal Comment 13:

"The inclusion of a large body of data from edge-of-field experiments such as the Johnson and Baker 1982 and 1984 studies in Table 10 of the PD-1 is also misleading. These experiments represent worst case estimates of alachlor entering small streams, and do not provide a measurement of ambient surface water concentrations."

Agency Response:

Johnson and Baker's 3-year study of field-to-stream transport of chemicals from Four Mile Creek in Iowa was not designed to provide ambient water quality monitoring data for alachlor, but to provide information needed for calibration and validation of predictive mathematical models. It is not misleading as alleged by Monsanto. It is possible that the 268 ppb of alachlor measured in water from Four Mile Creek resulted from a high intensity, large volume rainfall event which caused runoff from an alachlor-sprayed field.

Rebuttal Comment 14:

"A large number of positive findings are in the 0.001 to 10 ppb range and are unconfirmed by GC-MS. Monsanto has reviewed Baker's methods and has found no evidence of validation by recovery experiments below 2 ppb. Limited recovery experiments in the 2 to 10 ppb range would not support method detection limits of 10 ppb by an independent peer-review process, based on American Chemical Society and other guidelines for water monitoring and other environmental assays at such levels."

Agency Response:

The Agency understands that alachlor monitoring data were confirmed by two gas chromatographic columns, and not by GC-MS. The recovery and reproducibility are not accurate at low level measurements of alachlor. The precision of the monitoring data is acceptable, but the accuracy is questionable (Leovey, 1985).

Rebuttal Comment 15:

Two studies conducted in Maryland (Wu, 1980 and Wu et al., 1983) were not reported in table 10 of PD-1. "In the first study, the highest concentration of alachlor measured was 2.9 ppb, with most measurements below 1.0 ppb. In the latter study, runoff samples were taken over a 3-year period from eight experimental watersheds. A single sample showed a flow-weighted alachlor concentration of 6 ppb, with 22 other samples taken year round showing less than 1 ppb alachlor."

Agency Response:

It is true the data from the two studies by (Wu, 1980 and Wu et al., 1983) are not included in Table 10 of PD-1. The levels of alachlor residues in the runoff waters are stated correctly. The alachlor analyses were done by GLC only, and confirmation of alachlor was not conducted with mass spectroscopy. Since these data were not confirmed by GC-MS, nor the detection limits reported, the accuracy is questionable; measurement of low levels of alachlor are therefore underestimated.

Rebuttal Comment 16:

Surface water modeling (HSPF) issues raised by Monsanto relating to the use of the HSPF model are: (1) model time step sensitivity; (2) extrapolation of the Four Mile Creek study to the Iowa River Basin; (3) adequacy of precipitation records used in alachlor modeling; (4) chemical application procedures; (5) adjustment of infiltration parameters for tillage practices; (6) calibration and validation of runoff/stream flow; (7) sediment simulation; and (8) comparison of simulated and observed data on small and large river basins.

Agency Response:

(1) Model Time Step Sensitivity

Monsanto states that the HSPF application for estimation of alachlor concentrations, as described in Mulkey and Donigian (1984), should have been run with a 5-minute time step based on a statement in the HSPF user manual. Since the simulations were performed with a 2-hour time step, they claim that the modeling results are incorrect, and cite some HSPF simulations they performed to demonstrate the sensitivity of the results to the choice of time step.

The choice of a simulation time step is an important consideration in watershed modeling. The time step selected must be consistent and compatible with the space scale of the simulation, the time discretization of the available input data, and the objective purpose of the modeling study. Experienced watershed modelers would agree that a 5-minute time step would be inappropriate for hydrologic and pesticide modeling of the 2700 sq. mi. Iowa River Basin. Moreover, the available precipitation data are only available at hourly and daily time increments.

Monsanto's use of HSPF to study the sensitivity of the model results to the time step is incorrect. They used Test Run #9 provided with the HSPF distribution tape; to analyze the differences in model predictions when the time step was changed from 1 hour to 5 minutes. However, when changing the time step, Monsanto apparently did not properly adjust the units for the infiltration parameter. They used a value of 1.56 in/hr for the 5-minute model run as compared to a value of 0.13 in/hr in the 1-hour model run. This error caused the dramatic differences in selected model results and thus invalidates their conclusions; there was more than an order of magnitude difference in the infiltration rates they used in the two model runs.

Monsanto references a statement in the HSPF user manual on the documentation of the MSTLAY module that warns the user to employ extreme caution when using the module on anything but a 5-minute time step. This statement is both incorrect and outdated. The Agricultural Run-Off Model (ARM) model from which the MSTLAY module was developed uses 5 minute, 15 minute, and hourly time steps, not just 5-minute time steps. Also, simulations in the Four Mile Creek study (Donigian, et al, 1983) and Iowa River HSPF applications (Imhoff, et al, 1983; Mulkey and Donigian, 1984) used 15-minute, hourly, and 2-hour time steps with reasonable results. Thus, the 2-hour time step used in the HSPF alachlor simulations is both appropriate and justifiable.

(2) Extrapolation of Four Mile Creek Study to Iowa Basin

Monsanto claims that the alachlor predictions in the Mulkey and Donigian report are overestimated because of the lack of alachlor data in the Iowa River for calibration purposes, and the subsequent use of parameters from the Four Mile Creek study.

The extrapolation of parameters developed on a small watershed to a much larger river basin is a valid technical issue. The Four Mile Creek and Iowa River applications of HSPF are the only studies, to the Agency's knowledge, where pesticide modeling on a small area was used to extrapolate to modeling a much larger area. However, this type of extrapolation is standard procedure for hydrologic modeling and has been used for the past 20 years; similar procedures have been used successfully in nonpoint water quality modeling for BOD, sediment, nutrients, etc. (Hartigan et al., 1982, Southerland et al., 1982; Gilbert et al., 1982).

In the Four Mile Creek and Iowa River studies, the good simulation of the May 29, 1976, storm on Four Mile Creek led to the surprisingly close predictions for the same storm on the Iowa River. This ia a reasonable confirmation that the extrapolation from the small watershed to the larger river basin is valid for pesticide simulation.

(3) Adequacy of Precipitation Records Used in Alachlor Modeling

Monsanto charges that the use of precipitation data in the Iowa River simulations was unrealistic, was "designed to produce the most severe predictions" of runoff and alachlor concentrations, and was meant to represent a 'hypothetical' condition, not a realistic representation of runoff alachlor transport and behavior.

Three separate precipitation gauges were used in the Iowa River calibration/verification to represent precipitation on the northern, central, and southern portions of the basin. Adjustments to these gauges were performed based on standard hydrologic procedures that have been used in watershed modeling for the past 20 years.

The three gauges were selected to provide a reasonable representation of rainfall variability across the watershed and because the data were readily available in an HSPF database used in the earlier study of the Iowa River. Timing and resources for the study precluded the development of any additional rainfall time series for use in the modeling. Moreover, to the Agency's knowledge, there are no studies that indicate that the use of one rain gauge will necessarily produce more runoff than the use of multiple gauges, unless the chosen gauge has more rainfall volume and/or higher intensity characteristics than the others.

Consequently, there is no a priori reason to infer that the use of other precipitation data would lead to alachlor concentrations significantly different than those predicted by Mulkey and Donigian.

(4) Chemical Application Procedures

Monsanto questions the validity of the procedures used to specify the rate and timing of alachlor applications. They claim that the procedures of dividing the total application amount into increments of 25 percent, 50

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percent, and 25 percent applied on three separate dates within a 15 to 20 day period are unrealistic, and are not representative of any general agricultural practice of alachlor.

This is a legitimate question. However, it has been shown, based on the simulation results, to work reasonably well in both Four Mile Creek and Iowa River.

(5) Adjustments to Infiltration Parameters

Monsanto questions the procedures for adjusting the HSPF infiltration parameter (INFILT) to reflect the impacts of frozen ground conditions and tillage practices. They further charge that they have been prevented from rebutting this point because no material was provided in response to their data requests to allow Monsanto to determine the basis for the changes.

However, in the Mulkey and Donigian report, on the same page cited by Monsanto, it clearly states that the adjustment procedures were derived from the original Iowa River Study (Imhoff et al., 1983). Also, the Mulkey and Donigian report explicitly draws heavily from the Four Mile Creek Study (Donigian et al., 1983) which is referenced throughout the report. Both of these references are public information.

(6) Runoff/Streamflow Calibration and Validation

Monsanto criticizes the HSPF hydrologic/hydraulic calibration and validation primarily in terms of the presentation of data and model results, and the use of statistical model performance measures. They claim that statistical measures were used incorrectly and that the model validation was not successful.

The performance of a watershed model must be based on numerous comparisons of observed and simulated values, including the following:

- Daily flow values
- Monthly and annual runoff/flow volumes
- Daily cumulative flow frequency

Mulkey and Donigian performed all these comparisons in the HSPF hydrologic simulation of the Iowa Basin, and used regression statistics and other statistical tests as performance indicators. No single measure, test, or comparison is universally recognized as a criterion for judging model performance. The end result of the comparisons was that the HSPF simulations provided a fair and reasonable representation of the hydrologic response of the Iowa Basin, especially considering the numerous data problems and deficiencies. It was judged that the results were adequate for performing alachlor simulations as part of the surface water exposure assessment.

(7) Sediment Simulation

Monsanto's criticism of the sediment simulation results is similar to their criticisms of the hydrologic results. They state that the calibration and verification periods were inappropriately combined, and imply that this was done to minimize any apparent differences in the observed and simulated sediment loads.

In their criticism of the sediment simulation results, Monsanto failed to take note of the very good simulation of sediment concentrations shown in the Mulkey and Donigian report. In any case, differences in the sediment simulation will not have a significant impact on the predicted alachlor concentrations because alachlor is transported primarily in dissolved form.

(8) Comparison of Simulated Alachlor Results to Observed Data

The Monsanto discussion relating to the agreement between the predicted alachlor concentrations and the limited observed data is primarily a restatement of the information contained in the PD-1 and Mulkey and Donigian report, in slightly different form. However, Monsanto reaches the diametrically opposite conclusion from that of the Agency; they conclude that the modeling results are not consistent with the observed data, and that the model consistently over-predicts concentrations.

Since this appears to be a case where two groups review the same data and reach opposite conclusions, there is no need to restate the supporting arguments from PD-1 and the Mulkey and Donigian report. However, correcting an error in the PD-1, does serve to weaken Monsanto's arguments. In Table 10 in the PD-1, the peak alachlor concentration in Four Mile Creek, Iowa, on May 29, 1976 should be 96.0 ppb, not 9.60 ppb. This order-of-magnitude difference is important because the 1976 peak concentration is now significantly more consistent with the 1979 and 1980 peak concentrations of 267.7 ppb and 208.7 ppb, respectively, observed on the same watershed. This higher value is also more consistent with many of the other peak values noted in Table 10 of PD-1 (e.g., Honey Creek, Ohio; Sandusky River, Ohio; Wolf Creek, Ohio; and Chariton River, Iowa).

Monsanto's arguments are further weakened because the 1978 peak value of 54.5 ppb (Table 10, PD-1; p. 115, Vol. IV) should be footnoted to indicate that two storms in May and June 1978, were not properly sampled to identify a peak concentration. Two reports both Johnson, 1982 and Donigian et al., 1983, describe the situation that leads to missing samples for the events on May 27 and June 1, 1978. Because of the similarity between the May 29, 1976 storm and the May 27, 1978 storm in terms of timing, flow peak, and flow volume, (Donigian et al., 1983) calculated peak concentrations of about 130 ppb and 60 ppb for the missing May and June 1978 storms. Thus, the actual 1978 peak alachlor concentration would likely exceed 100 ppb and further strengthen the consistency with the other peak values.

These corrections improve the consistency and agreement between the data and model results, and thereby negate Monsanto's statement that the model results are not "reasonably consistent."

Monsanto's criticism of the model results for the May 29, 1976 storm on the Iowa River are unfounded. The comparison of the model predictions with the grab sample data for this storm was, in effect, a "blind" validation of HSPF because the data were not discovered until after the model runs were in progress. The comparison shown in the Mulkey and Donigan report does indeed demonstrate a "striking" agreement between the model results and the observations; even Monsanto's table shows that the peak concentrations agree within a factor of 2 to 3 at Marshalltown and Marengo. The observed data points (2 values) at Rowan do not record a comparable peak concentration; however, there is no reason to believe that a grab sample procedure for a runoff event will necessarily capture the peak concentration. Moreover, it is highly likely that the recorded peak values of 33 and 35 ppb near Marshalltown and Marengo, were not the actual peak values that occurred. The higher values simulated by HSPF may be more appropriate estimates of the actual peak concentration.

2. Applicator Exposure

Rebuttal Comment 1:

The reasoning for Monsanto's decision to remove aerial application from the alachlor label and the effect this decision may have on the aerial application industry was questioned.

Agency Response:

Aerial application was voluntarily removed from the label by Monsanto prior to the issuance of the PD-1. The Agency is proposing to reinstate aerial application on the label based on a review of published aerial exposure studies. This review indicated that per pound of active ingredient applied, pilots receive less exposure than do most ground boom applicators. Aerial and ground mixer/loaders would receive identical exposures per pound of active ingredient handled. In addition the Agency is proposing the addition of the following label restrictions: "Aerial application may be performed using mechanical flaggers only."

III. BENEFIT ANALYSIS

A. Introduction

Alachlor (Lasso®) is an acetanilide herbicide registered for control of many annual grassy weeds and certain annual broadleaf weeds before they emerge from the soil. Alachlor is registered for use on the following sites: corn (field, sweet, pop, seed), soybeans, peanuts, dry beans, lima beans (green), mung beans, green peas (processing), cotton, grain sorghum, sunflowers, and certain ornamental plants. Approximately 94 percent of alachlor usage is on three sites: corn (63 percent), soybeans (28 percent), and peanuts (3 percent). Total usage of alachlor in the United States is estimated at 80 to 84 million pounds active ingredient per year (Table 17). Alachlor is a major herbicide on several of its use sites. For example, it is used on more than one-third of U.S. field corn, one-fifth of the soybeans, and nearly two-thirds of peanuts.

The Agency has conducted an analysis of the benefits of alachlor, which is summarized in this section. The analysis describes the benefits which would be foregone to users and others affected if alachlor were cancelled for use on each of its registered use sites. Such an assessment provides a baseline estimate of the value of alachlor as currently used and registered to the agricultural community. It also provides a worst case assessment of the economic impact if all alachlor uses were to switch to alternative pest control measures as a result of the Agency's proposed regulatory action, an outcome the Agency believes is extremely remote. On the contrary, as indicated in the discussion below, the Agency believes that its proposed regulatory action will have minimal impacts on the use and value of alachlor, and that the loss to the agricultural community would be significantly less than if alachlor were unavailable.

Information used in the analysis was derived from several sources: scientific literature and weather data obtained from the National Agricultural Library, United States Department of Agriculture (USDA); Current Research Information System - USDA; published state recommendations; seed companies; PD-1 comments; company submitted data; various USDA reports; and Agency cooperative agreements with state universities (University of Georgia and University of Illinois).

The general approach was to collect and review relevant biological information and current research, and conduct a biological analysis (Schneider, Petrie, and Reese, 1986). This document is the basis for all biological data and assessments reported in this summary. The results of the biological analysis were provided as input to the economic analysis of alachlor benefits. Based on the biological parameters, the economic impacts of an alachlor cancellation were then estimated (U.S. EPA, 1986).

Table 17. Summary of Current U.S. Alachlor Use, by Crop

Use Site	Acres Planted	Acres Treated	Pounds ai Applied	Percent of Crop Treated	Percent of Alachlor Use on Crop
		(expressed in thousands)			
Corn(field)	74,697	25,500-26,500	50,519-52,500	35	63
Soybeans	64,918	13,000-14,000	22,052-23,748	21	28
Peanuts	1,400	904	2,790	62	3
Cotton	10,900	29-30	43-45	<1	<1
Dry Beans	2,000	230	508-623	15	<1
Grain Sorghum	15,806	1,400	3,275	8	4
Green Peas (processing)	299	6-9	13-20	2-3	<1
Lima Beans (green)	65	6-9	13-20	9-14	<1
Sunflowers	3,306	70	227	2	<1
Sweetcorn and Popcorn	1,200	300	600	25	<1
Ornamentals	N.A.	nominal	nominal	N.A.	N.A.
Total		41,445-43,452	80,040-83,848	N.A.	100

Cancellation of all uses of alachlor would be expected to result in first year losses at the farm level of $510 to $758 million, which represents both increased costs of weed control and decreased value of production (Table 18). These losses, while large in absolute terms, are relatively small compared to the overall value of affected crops. The impacts of alachlor cancellation need to be put into perspective of the current situation for agriculture. A substantial portion of farmers producing field crops are undergoing financial stress. The Food Security Act of 1985 (1985 Farm Bill) provides for record subsidies in an attempt to aid farmers. Cancellation of alachlor would be offsetting a portion of these subsidies and would therefore have the effect of decreasing the cost to society as a whole. The impacts of cancellation on alachlor users are more significant, as described in more detail below. In calculating these impacts, it was assumed that only currently registered chemicals would be available at the time of an alachlor cancellation. Alternatives to alachlor were chosen on the basis of comparative product performance, market availability and cost. Metolachlor is the principal alternative which currently costs slightly more than alachlor on a per pound active ingredient basis, but generally costs slightly less on a per acre treated basis. Also, the same yield will not be obtained with metolachlor in all cases as discussed later in this section.

It is projected that the burden of the loss of alachlor would be largely borne by the farmer and about one-third of the burden would be shifted to the consumer. In addition, cancellation could result in decreased exports of agricultural products. The overall loss to society would range from about $302 to $508 million the first year. Net overall losses would be nominal to consumers of food products affected, although the impact on individual farmers could be significant.

B. Analysis for Individual Commodities

1. Corn

The largest use of alachlor is on corn, which represents 63 percent of the total annual usage. Alachlor is used to control annual grassy weeds and certain broadleaf weeds on about 26,000,000 acres of corn or about 35 percent of the U.S. corn crop. Alachlor has been widely used on corn for approximately 16 years. Nearly 300,000 farmers currently use alachlor on corn acreage.

Acetanilides (e.g., alachlor and metolachlor) play a major role in corn/soybean rotations and in conservation tillage. Because acetanilides are not phytotoxic to rotated crops and are surface applied, they impose minimum disturbance of crop residues on the soil surface. Use of these herbicides permits implementation of conservation tillage, i.e., maintenance

Table 18. Summary of Benefits of Alachlor, by Site

Use (Site/ Site Category)	Key Pests	Avail. of Economic Alternatives (Qualitative Assessment and Likely Chemicals)	Economic Impact of Cancellation		
			Type	Extent	Significance
			(Qualitative Keyword Characterization)		
Field corn	Annual grasses	Alternatives available and in use. (Metolachlor-close substitute; also other chemicals and cultivation available.)	User:		
			Increased cost of production and yield losses.	Average yield losses 4.8 bu/acre (range: 3.9-7.3) in the first year. Will decrease to about 1 bushel (less than 1% of yield) after 10 yrs. First year impact of $318.1-$552.4 million to users.	Most likely first year losses of $1,680 to $1,800 per farm which could be quite important to some farmers. Would reduce to about $360 per farm after 10 years.
			Corn Prices	Less than 1-2% increase	Could reduce loss somewhat but would largely be offset by reduced government payments.
			Consumer:		
			Price increases possible in longer term.	Increases, if they occur, would be relatively small; 1-2% at maximum for corn based products.	Overall not very important.
			Market, Social:		
			Hasten dislocation of farmers, would hasten decline of small farm communities.	Hasten decline of small farm communities.	Overall, quite nominal, could be significant to some communities.
			Government Payments	Decrease $94-$246 million in early years.	Reduced. Less than 1% of expected 1986 farm program cost.
Soybeans	Annual grasses Nightshade Sicklepod	Alternative metolachlor produces comparable control under most conditions (sicklepod serious problem for some areas in South).	User:		
			Production cost increases	$113 million.	Overall, not very important. Losses of $1,400 per farm which could be important to some farmers due to current financial stress.

Table 18. Summary of Benefits of Alachlor, by Site (cont'd)

Use (Site/ Site Category)	Key Pests	Avail. of Economic Alternatives (Qualitative Assessment and Likely Chemicals)	Economic Impact of Cancellation		
			Type	Extent	Significance
			(Qualitative Keyword Characterization)		
Soybeans (cont'd)			Yield losses	Yield loses of 3.5 to 5 bushels; $40.6 to $46.5 million.	
			Prices	Up less than 1%	Price increases would not offset reduced yields for users. Non-users would have revenue gain.
			Consumer:		
			Price increases for soybean products possible.	Less than 1%.	Overall, not very important.
			Market, Social:		
			Hasten dislocation of farmers with accompanying impacts on rural communities.	Small portion of users.	Overall, not important. Could be severe in some communities.
			More difficult to compete in world markets	Exports down less than 1%.	Not major factor.
Peanuts	Annual grasses Sicklepod Florida beggarweed	Chloramben and metolachlor.	User:		
			Little direct impact except those with sicklepod, beggarweed problem.	Yield losses in Southeast (13-15%) and Southern Plains (9%). Up to $35 million net loss to users. Windfall gain to nonuser of about $15 million.	Moderate.
			Revenues	Up about $37 million.	Would shift most of impact to consumer.

Table 18. Summary of Benefits of Alachlor, by Site (cont'd)

Use (Site/ Site Category)	Key Pests	Avail. of Economic Alternatives (Qualitative Assessment and Likely Chemicals)	Economic Impact of Cancellation: Type	Economic Impact of Cancellation: Extent	Economic Impact of Cancellation: Significance
			(Qualitative Keyword Characterization)	(Qualitative Keyword Characterization)	(Qualitative Keyword Characterization)
Peanuts (cont'd)			Consumer:		
			Price increase	About $37 million.	Not very important (2% of value of peanut crop).
			Market, Social:		
			No significant impacts expected.		
			Government payments	Little extent.	Would adjust quotas and increase prices to consumers.
Dry beans	Annual grasses Nightshade	Metolachlor a close substitute.	User:		
			Cost of production increases as value of production decreases.	Loss of up to $12.75 per acre for 15% of total dry bean acres.	Overall not important; represents less than 1% of overall value of production. Could adversely affect some users.
Others	Annual grasses	Several effective alternatives available.	User:		
			Mostly negligible; significant cost increases and yield losses possible for sweet and popcorn.	$3.0-$12.3 million.	Negligible except for sweet and pop corn. Could adversely affect some users.
All uses	Annual grasses Sicklepod Nightshade	Economic alternatives available for most sites; metolachlor close substitute, along with others.	User:		
			Production cost increases and yield losses.	Initial year impacts would be $510-$758 million. Will decrease over time.	Minor compared to total farm income (2%-4%) initially, less than 2% in long-run. Some users adversely affected in short-run partially due to financial crisis in agriculture.

Table 18. Summary of Benefits of Alachlor, by Site (cont'd)

Use (Site/ Site Category)	Key Pests	Avail. of Economic Alternatives (Qualitative Assessment and Likely Chemicals)	Economic Impact of Cancellation		
			Type	Extent	Significance
			(Qualitative Keyword Characterization)		
All uses (cont'd)			Consumers:		
			Food prices	Increases less than 1%.	Nominal.
			Market, Social:		
			Crop prices	Less than 1%.	Nominal.
			Ag. program costs	Down by $95-$246 million first year.	Relatively minor.
			Efficiency loss	$302-$508 million.	Overall, probably not very important. Some effect on competition in world markets expected.

of 30 percent of crop residue on the soil surface. However, alachlor has an advantage over metolachlor, since less rainfall is required to activate alachlor.

Alachlor is utilized as part of a corn weed management system for control of annual grasses and broadleaf weed species including triazine resistant weeds. Other currently available herbicides that may perform those functions include pendimethalin, butylate, EPTC, high rates of the triazines (atrazine, simazine, and cyanazine), and metolachlor. All of these herbicides have various use limitations including soil, geographical, rotational, phytotoxic, and weed spectrum differences.

Pendimethalin must be soil applied to avoid corn injury, and rainfall is essential to its activation. Pendimethalin cannot be soil incorporated, has soil type restrictions, and it is less effective against several broadleaf species. Butylate and EPTC must be soil incorporated thus limiting their use in conservation tillage systems. Butylate is further limited in usefulness by its failure to control broadleaf weeds. Accelerated biodegradation (loss of efficacy) has been a problem with continuous use of the butylate and/or EPTC. Use of high rates of atrazine increases the potential for herbicide carryover to rotational crops under certain weather and soil conditions. The use of high triazine rates has been associated with the development of triazine resistant weed biotypes.

The primary alternative to alachlor is metolachlor. Although metolachlor can be considered comparable to alachlor as a herbicide in many ways, there are differences which could become more important if metolachlor were used as widely as alachlor. Metolachlor has more rate restrictions on its use and does not have the wide flexibility of rate variation to counter some specific weed problems. Alachlor has been found to control some broadleaf weeds better than metolachlor. Less broadleaf weed control from metolachlor could lead to the use of other herbicides, such as triazines. Increased triazine use or increased triazine rates may enhance development of triazine resistant weed biotypes. Annual grass control is generally comparable between alachlor and metolachlor. When comparing label and current recommendations, two sequential alachlor treatments are allowed for control of woolly cupgrass and shattercane grasses as opposed to only one treatment on the metolachlor label. Alachlor sequential treatments are currently used for control of these two grass species on approximately 520,000 acres, resulting in better weed control on these acres. Generally, the product performance analysis of alachlor showed that alachlor provides better weed control than metolachlor in high organic matter soils with a fine texture while metolachlor provides better weed control than alachlor in coarse mineral soils with low organic matter such as those common in the

Southern United States. Alachlor has also been shown to cause less stress than metolachlor on corn plants under cold and wet conditions. Available phytotoxicity/stress data and the difference in weed control indicate that yield losses from use of metolachlor rather than alachlor could average 4.8 bu/A with a range of 3.9 to 7.3 bu/A depending upon weed species, present hybrid tolerance, and weather conditions (U.S. EPA, 1986). Of this 4.8 bu/A average yield loss, the overall differential due to weed control equals approximately one bushel, with the phytotoxic/stress differential accounting for the remaining 3.8 bu/A. The weed control differential is highly concentrated in certain states and the phytotoxicity/stress effect in the northern corn growing areas of the United States.

The phytotoxicity/stress effect has developed over time in seed corn because of the early introduction and continued use of alachlor by the seed companies as a standard grass herbicide in their plots. As a consequence, the corn has become more tolerant to alachlor. Based on available seed company studies that compared herbicide performance, 70 percent of hybrids tested were more tolerant of alachlor than metolachlor. The other 30 percent of hybrids tested exhibited comparable tolerance to both alachlor and metolachlor. It is assumed that these data reflect the current hybrid tolerance situation in the North Central corn producing states. If alachlor were not available, it is expected that 5 to 10 years of dedicated seed corn screening and development could be required to produce a comparable amount of metolachlor tolerant seed (Schneider, Petrie, Reese, 1986).

The biological conclusions presented are based on yield data obtained from an analysis of the scientific literature and on yield data obtained from seed companies. Seed company studies of comparative herbicide performance are conducted to determine the sensitivity of their popular hybrid seeds to widely used pesticides. Maximum yield from their hybrids is the motive, not herbicide endorsement. The seed company corn hybrid tests are conducted throughout the North Central United States corn growing region and are representative of effects that could be expected in the most predominant alachlor usage area of the United States. Data utilized for conclusions reflect recommended rates and current agronomic/cultural practices. Data points evaluated by most seed companies are well defined, allowing detailed evaluation of most field variables (Schneider, Petrie, Reese, 1986).

Data submitted and/or referenced by Monsanto Agricultural Products Company and Ciba-Geigy Chemical Company were useful for their intended purpose; to document efficacy claims for initial product registration. However, these data were not considered to be representative of the comparative performance of alachlor for the following reasons:

- Test protocols and rating methods were not consistent for Monsanto Ciba-Geigy USDA/ University generated test data. Test protocols used for company generated data may tend to support the company position.

- These field tests were generated to establish product efficacy and were not designed to evaluate comparative product performance over a period of years in the same locations to determine differential yield loss.

- The majority of these tests are dated and fail to reflect current formulations used, and current agronomic/cultural practices such as "surface blend", conservation tillage, corn/soybean rotation, current hybrids.

- Hybrid sensitivity could not be determined, as required data points were not collected (i.e., stand reduction, emergence counts, stand heights, barren plants, and percent lodging). These tests were not designed to scrutinize yields beyond weed control. Confounding test variables that affect ratings and yields were not explained. in most tests.

- Weather variables were not described nor accounted for in most tests.

Other data that were not utilized for the final biological conclusions include certain PD-1 rebuttal statements that provided no description of test parameters, hybrids, weed infestations, weather variables and other weighting factors; and certain scientific literature citations that were deficient for the same reasons (Schneider, Petrie, Reese, 1986).

Metolachlor was assumed to be the alternative for alachlor under most situations, with additional herbicides and/or cultivations used under some conditions. Metolachlor substitution could increase average weed control costs by about $1.50 per acre for alachlor users. Estimated yield losses of 3.9 to 7.3 bushels per acre with an average loss of 4.8 bushels per acre would occur initially. These yield losses would be mainly in the major corn producing states in the North Central Region of the country. These losses could decrease to about one bushel per acre after about 10 years.

Partial budgeting analyses and econometric analyses were used to examine the impacts of an alachlor cancellation. A cancellation of alachlor use on corn is estimated to result in annual losses to users of $318.1 to $552.4 million the first year following a cancellation with a most likely loss of about $388 million (U.S. EPA, 1986). The estimated losses include decreased deficiency payments (government subsidies) which could not occur the first year since deficiency payments are based on historical yields rather than current yields. Increased production costs range from $40.6 million to $42.5 million, value of production losses range from $143.6 to $335.3 million, and decreased deficiency payments of $94.4 to $246.1 million. These losses in aggregate represent about three percent of expected value over all corn production and about $1,150 to $6,462 for the typical alachlor user with 120 acres of corn. First year user losses could range from about $9.60 to $54.00 per acre with most likely losses being $14.00 per acre which represents about 20 percent of typical returns of about $70.00 per acre. These losses are enough that they could hasten the exit of some farmers from agriculture.

User losses could decrease to $75 million after metolachlor tolerant corn fully penetrated the market over about a ten year timespan. Production costs are estimated to decrease by about $21.8 million and value of production losses range from $50.3 to $63.4 million with decreased deficiency payments of $33.4 to $46.6 million. After such adjustments, per acre losses would range from about $1.00 to $33.00 with an average loss of about $3.00 or 4 percent of expected net returns. These losses in aggregate represent about one percent of expected value of overall corn production and about $360 for the typical alachlor user with 120 acres of corn. These impacts would be large enough to force a shift of a portion of corn acres out of corn production and into other uses of the land. Due to the nature of the current farm subsidy programs, if corn price increases are sizeable due to decreased yields, any increased price of corn products could be largely offset by decreased deficiency payments. This implies that corn growers who currently do not use alachlor could have increased revenues from higher corn prices and decreases in deficiency payments largely offsetting increased revenues. In addition, consumers as a group would not experience significant impacts since increased corn prices could be largely offset by decreased cost of agricultural programs.

Exports of corn and corn products could decrease slightly (by less than one percent) due to the projected increase in corn prices which would make U.S. production less competitive on world markets.

Other studies have examined the impacts of an alachlor cancellation on corn and soybeans. Monsanto, as part of their response to the alachlor PD-1, provided an economic analysis on the potential impacts of a cancellation (Monsanto, 1985). The analysis was developed by an independent economic consulting firm and used Monsanto supplied data. The Monsanto

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analysis estimated that the Gross National Product would decrease by $1.3 billion the first year after a cancellation of alachlor. In arriving at their estimated impacts, Monsanto included effects on their employees as well as overall multiplier effects. The Agency does not include impacts on chemical firms, chemical firm employees, or multiplier effects in its benefits analyses. Finally, the Monsanto analysis could not consider changes implemented in the 1985 Farm Bill which was enacted into law after the due date for their comments.

The Department of Agriculture (USDA) published a preliminary analysis of economic implications of potential cancellations of corn and soybean herbicides (Osteen and Kuchler, 1986). The USDA results are preliminary and are not an official response by the Department of Agriculture to the PD-1. USDA's analysis was based on expert opinions of corn and soybean specialists from the major corn producing states. While this publication is preliminary and does not include implications of the 1985 Farm Bill, the annual welfare loss from a cancellation of alachlor use on corn and soybeans was estimated at about $124 million the first year and $149 million after adjustments. The Agency position is that the efficiency loss from a cancellation of alachlor (approximately the same measure as used by USDA) would range from $243 to $441 million the first year after cancellation. The first year estimates are considerably different ($124 million vs. $285 to $469 million). Further, the USDA estimated the annual impacts of a cancellation for both alachlor and metolachlor to be about $2.12 billion. The impact estimates from the USDA analysis were not specifically used by the Agency in its calculation of impacts because published data indicated that yield differences between alachlor and metolachlor exceeded those used by the USDA in their economic analysis and the Agency attempted to capture changes brought on by the 1985 Farm Bill.

2. Soybeans

The second largest use of alachlor is on soybeans, which represents 28 percent of the total annual usage. Alachlor is used to control annual grassy weeds and certain broadleaf weeds on about 13.5 million acres of soybeans or about 21 percent of the U.S. soybean crop. Nearly 200,000 farmers currently use alachlor on soybean acreage.

As shown in the Product Performance Evaluation of Alachlor (Schneider, Petrie, Reese, 1986) soybean weed management systems utilize alachlor to control annual grasses and problem broadleaf weeds. Herbicides that may be partial substitutes for alachlor include the dinitroanilines (trifluralin, pendimethalin, oryzalin, ethalfluralin), vernolate, and metolachlor. The dinitroanilines do not control some of the major broadleaf weeds as well as alachlor and are not labelled for use on high organic soils. Metolachlor is the primary alternative to alachlor in soybeans and is comparable under most conditions. However, alachlor has provided better broadleaf weed control in the North Central Region and has shown better sicklepod control in the Southern U.S. Alachlor performs well in high organic soils areas of Virginia and North Carolina where metolachlor cannot be used. Without alachlor, additional herbicide treatments would be required for broadleaf weed control and in organic soils two additional cultivations would be necessary. Even with these measures, yield losses in certain areas in the absence of alachlor could range from 3.5 to 5.0 bu/A.

Metolachlor would be the primary substitute for alachlor on soybeans with additional herbicides and/or cultivations used under some conditions. Under some conditions, farmers may employ other herbicides which were not explicitly considered in this analysis. The use of alternatives could increase average weed control costs by about $8.00 per acre for alachlor users. Yield reductions of 3.5 to 5 bu/A could occur on about 18 percent of those acres currently treated with alachlor. These losses would be mainly in the Southeast.

Partial budgeting and econometric analyses were used to estimate the impacts of a cancellation of alachlor on soybeans. The cancellation of alachlor use on soybeans would result in annual losses to users of $153.6 to $159.5 million (U.S. EPA, 1986). This represents increased production costs of $113 million and decreased value of production of $40.6 to $46.5 million. These losses in aggregate represent less than two percent of the expected value of soybean production and about $1,400 to the typical soybean producer. User losses would range from about $3.00 to $37.00 per acre with average losses being about $11.50 per acre. The cancellation of alachlor use could result in a reduction in net income of about 7 percent to over 100 percent.

This is expected to accelerate the decrease in soybean production which will occur under current market conditions. An econometric analysis conducted for the Agency indicated that a portion of soybean acres in the southeast would go out of soybean production and a high portion of those going out of soybeans would go out of agriculture. This largely represents a shift back out of crop production acres which came into production during the boom years of the 1970's with the related impacts on the individual producers.

These impacts lead to increased soybean prices which could result in windfall gains for non-users of alachlor as well as increased costs of soybean products for the consumer. Costs to consumers for soybean products are expected to increase less than one percent. Exports of soybeans could decrease slightly (less than one percent) due to the increased prices for soybeans making U.S. production less competitive on world markets.

Other studies have examined the impacts of an alachlor cancellation on soybeans and are discussed in the corn section.

3. Peanuts

Alachlor is an important part of a weed management system in peanuts. Over 90 percent of the alachlor which is used on peanuts is applied as a cracking stage treatment. Metolachlor is the only herbicide that could be used as a substitute. However, alachlor gives better control of broadleaf weeds, such as sicklepod and Florida beggarweed in the Southeastern peanut growing region, than metolachlor. Most of the peanut acreage would require additional postemergence herbicide treatments if metolachlor were used in lieu of alachlor. In addition, peanut yield losses of 13 to 15 percent could occur in the Southeastern region if alachlor were not employed.

Metolachlor and chloramben are the herbicides which were assumed to be used most commonly to replace alachlor on peanuts. Additional cultivations would be employed under some situations. Yield reductions of 13 to 15 percent could occur on a large portion of the acres in the Southeast and 9 percent could occur on a large portions of the acres in the Southern Plains. Alachlor is used on about 900,000 acres of peanuts which represents about 60 percent of U.S. planted acres.

The cancellation of alachlor could result in user losses of about $35 million (U.S. EPA, 1986). These losses represent about two percent of the expected value of peanut production and about $2,000 to the average peanut producer. On a per acre basis, income could be reduced by about $38.70, which represents about 12 percent of expected income of $300.00. It was estimated that consumers could have losses of about $37 million. This was based on an estimated overall loss of about $57 million of which about two-thirds would be shifted to the consumer through passing increased costs of production to meet quota as provided for in the 1985 Farm Bill. Non-users could have a windfall gain of about $15 million. The remainder of production would be for export (it was assumed that farmers would bear this loss since this impact on total U.S. peanut production represents less than ten percent of world peanut production).

4. Dry Beans Including Red Kidney Beans and Mung Beans

Dry bean production is concentrated in the Western U.S. States, Michigan, and New York. Dry beans represent less than 1 percent of the total alachlor usage with about 15 percent (about 230,000 acres) of the crop treated for control of annual grassy and broadleaf weeds such as nightshades. The loss of alachlor would result in annual losses of $0.4 million to $1.5 million with the average being 0.9 million. These losses represent increased production costs to alachlor users. On a per acre basis, production costs could increase by up to $12.75. Impacts could be concentrated in the states of Michigan, Nebraska and Colorado. Nightshades are troublesome in dry beans because they reduce yields, are poisonous to livestock and humans, lower quality of the beans, and impede harvesting. In California, equally effective alternatives for alachlor include ethalfluralin EPTC, and metolachlor for hairy nightshade control. In Idaho, ethalfluralin is reported to be superior to both alachlor and EPTC. However, in Washington, metolachlor can carryover to subsequent crops while alachlor does not injure rotated crops. In Michigan, metolachlor is 15 to 20 percent less effective than alachlor in control of black nightshade. Without alachlor, dry bean growers would either incur additional dockage for nightshade stained beans at the elevator or hand rogue nightshade plants before harvest (Schneider, Petrie, 1984) and (Schneider, Petrie, Reese, 1986).

5. Other Sites Including Grain Sorghum, Sweet Corn and Popcorn, Sunflowers, Cotton, Lima Beans (Green), Green Peas, Ornamental Woody Plants

Information on these sites was evaluated (Petrie and Schneider, 1984[a,b]; Schneider and Petrie 1984[b,c,d]) and indicates that these sites account for less than 1 percent of the total alachlor usage. Information on alachlor use for each of these sites was rather limited compared to those crops discussed previously. Therefore, information presented here is subject to greater uncertainty than for other uses. In most cases, except sunflowers, metolachlor is an alternative to alachlor but with differences in broadleaf control and price.

a. Grain Sorghum

Alachlor is used on about 1.4 million acres of grain sorghum which represents about 8 percent of U.S. planted acres. It was estimated that the cancellation of alachlor registrations for use on grain sorghum would have little if any impacts to users. Metolachlor, the currently registered alternative, appears to be the preferred herbicide for use on grain sorghum with little difference between the two materials.

b. Sweet Corn or Popcorn

Current information indicates that about 300,000 acres of sweet corn and popcorn are treated with alachlor which represents about 25 percent of planted acres. It was assumed that factors that affect field corn would affect sweet corn and popcorn in a like manner. Metolachlor would be the primary alternative herbicide to alachlor on these crops with additional herbicides and cultivations used under some conditions. However, these crops are of higher value than field corn with equal sweet corn/popcorn yield reductions having a larger impact on the farmer than equal yield reductions on field corn.

Partial budgeting was used to estimate the impacts of a loss of alachlor on sweet corn and popcorn. The cancellation of alachlor registrations for sweet corn and popcorn could result in losses of $3 to $10 million to impacted farmers. Weed control costs could increase about $300,000 with value of production losses of $2.8 to $9.6 million. On a per acre basis, this represents losses per farmer of about $9.00 to $32.00. Information available indicated that the losses would be borne largely by the farmer with little passed on to the consumer.

c. Sunflowers

Less than 1 percent of the sunflower (70,000 acres) crop is treated with alachlor for weed control. The loss of alachlor could result in annual minor losses. Alachlor is useful in sunflowers grown under conservation tillage. Alternatives to alachlor include trifluralin, EPTC, and pendimethalin.

d. Cotton

Alachlor is currently used on about 30,000 acres of cotton, which represents less than one percent of the U.S. planted acres. Alachlor is currently registered for use only in Oklahoma and Texas. The cancellation of alachlor use on cotton could result in losses of about $160,000 to users. Alachlor is recommended for control of annual grasses. However, it has a narrow margin of crop safety (phytotoxicity) on cotton. Alternatives include metolachlor and trifluralin.

e. Green Peas (for processing)

Alachlor is registered for use on green peas in Minnesota only. Usage appears extremely limited. Metolachlor has comparable grass control, while alachlor and propachlor have an added advantage in that they can be used on higher organic soils.

f. Lima Beans

Alachlor is currently used on about 6,000 to 9,000 acres of Lima beans (green). This represents about 10 to 14 percent of U.S. planted acres. The cancellation of alachlor would result in little, if any, efficiency losses to society.

g. Ornamentals

Alachlor is registered for use on selected woody ornamentals. The Agency was unable to find any indications of use of alachlor on ornamentals.

C. Analysis of Rebuttal Comments Concerning Benefits

Alachlor PD-1 rebuttal statements were reviewed for information regarding the current benefits derived from previous and current use of alachlor, alachlor use in relation to crop growth and tillage practices, comparative product performance with alternatives and proposed regulatory options. Just under 1,000 rebuttal letters were received, approximately one-quarter of which were form letters. Many of the rebuttals from USDA Scientists, university scientists, seed companies, individual growers and grower groups were helpful in identifying specific alachlor benefits.

To briefly summarize the rebuttal comments received claim that alachlor herbicide has been used effectively and consistently for 16 years by some growers as part of their crop management system; with no evidence of weed resistance, accelerated microbial degradation significant injury to treated crops, or carry-over injury to fall-seeded or spring-rotated crops. They state that alachlor is not limited by a narrow rate range, nor is it restricted to soils with a given pH or organic matter content. Alachlor can be used by a grower on numerous crops such as corn, soybeans, grain sorghum, peanuts or sunflowers on their farm. These crops can be grown using conventional tillage, conservation tillage, or no-tillage with no carry-over injury to these crops if rotated the next year. This versatility allows the grower to buy a limited number of herbicides in bulk well in advance of planting time. If weather conditions at the time of planting dictate a change in cropping pattern or application where alachlor is used, the grower can easily modify his schedule to compensate. Further, if a portion of his earlier planted crop is destroyed because of flooding or hail damage after alachlor use, the growers can replant with a short-season soybean or grain sorghum crop to recover their losses with no injury to the second crop. Alachlor users are pleased with the broad spectrum of annual grass and broadleaf weed control obtained including control of certain triazine-resistant weeds. Alachlor is often selected by growers whose fields have relatively high soil organic matter content. A recently registered use of two sequential applications of alachlor is utilized by some growers for control of troublesome grassy weeds such as woolly cupgrass and shattercane.

Chemical company rebuttals were received from the Monsanto Chemical Company, Ciba-Geigy Chemical Company, Stauffer Chemical Company, and the Elanco Chemical Company. Monsanto later provided the Agency with a computer tape that summarized the corn yield data. Ciba-Geigy Chemical Company provided references to efficacy data in EPA files. These data were submitted to EPA in the 1970's to Ciba-Geigy support the registration of metolachlor herbicide. Stauffer and Elanco chemical companies did not submit or reference product performance data but did submit PD-1 rebuttal statements. All company rebuttals received were considered in the development of the benefits assessment but did not raise any new issues.

Benefits data submitted by Monsanto and referenced by Ciba-Geigy consisted of mostly small plot side-by-side herbicide studies conducted by academia and by the chemical companies; some of which were carried to yield and few of which were statistically analyzed. The majority of these tests were conducted by company personnel while their products were undergoing experimental use or under the supervision of company personnel on farms. Monsanto submitted 115 University generated trials. From their data Monsanto concluded that a grower using Lasso would obtain an average of 5.9 bushels per acre more corn than if the same grower used metolachlor. One of the 5.9 bushels, Monsanto concluded that 4.0 bu/A was attributable to less phytotoxicity to the corn and 1.9 bushels/A was due to superior weed control. In soybeans, Monsanto concluded that a grower using alachlor would obtain three extra bushels of soybeans per acre than if metolachlor were used due to superior weed control. Less injury to rice rotated after soybeans was also cited as a superior alachlor benefit.

In peanuts, superior broadleaf weed control was cited by Monsanto over alternative herbicides.

Monsanto, as part of their response to the alachlor PD-1, provided an economic analysis of the potential impacts of a cancellation. The analysis was conducted by an independent economic consulting firm and used Monsanto supplied data. This analysis projected that the real output of the U.S. economy, as measured by gross national product, would be reduced by $19.1 billion (1984 dollars) over the 1986-1993 time period. The first year decrease in gross national product would be $1.3 billion. In arriving at their estimated impacts, Monsanto included effects on their employees as well as overall multiplier effects. The Agency does not include impacts on chemical firms, chemical firm employees, or multiplier effects in its benefit analyses. In addition, the Monsanto analysis did not consider changes implemented in the Food Security Act of 1985 (1985 Farm Bill) which was enacted into law after the due date for their comments. Finally, the Monsanto analysis estimated changes in gross national product which is a different measure than the efficiency loss estimates used by the Agency. Ciba-Geigy concluded from their data base that there is no "significant difference in yield between alachlor and metolachlor in comparative test plots."

After a careful review of the Monsanto and Ciba-Geigy data bases, the Agency concludes that the majority of these tests do not represent current formulations or current agronomic/cultural practices commonly used. Also, these tests are not cross-compatible because different protocols and rating systems were used by Monsanto, Ciba-Geigy, and USDA/University researchers. Corn hybrid sensitivity was not properly evaluated nor were all field variables reported in many tests.

In an effort to obtain more current information and to more carefully evaluate the corn hybrid sensitivity issue raised by Monsanto (Volume 24, PD-1 rebuttal), numerous seed companies were contacted and a literature search was conducted. Of the numerous corn hybrid seed companies contacted, three were currently conducting side-by-side herbicide screening trails in the field. Conclusions reached from an Agency review of these data are presented in Chapter III (Benefits Summary).

IV. REGULATORY OPTIONS AND RISK/BENEFIT ANALYSIS

A. Introduction

As previously explained, FIFRA requires the Agency to weigh the risks against the benefits for each use of a pesticide in order to determine whether continued registration would cause unreasonable adverse effects on the environment. In Chapters II and III, the risk posed by exposure to alachlor is assessed and the benefits of alachlor use are evaluated. The Agency has identified a number of regulatory options, and has evaluated each option for its impact on both risks and benefits.

In addition, the Agency has identified registered alternative pesticides for the various uses of alachlor. The general risks of alternative pesticides have been summarized based on the available data.

This section identifies the regulatory options available to the Agency to reduce the risks from the registered uses of alachlor. Each option has been evaluated for its impact on the risks and benefits of the registered uses of alachlor and the most appropriate regulatory options have been proposed.

B. Risk Conclusions

Exposure to alachlor can occur through application of the pesticide to agricultural and ornamental crops, harvesting the crops, and eating foods containing residues of alachlor. Exposure can also occur through contaminated drinking water. In these ways, the entire U.S. population may be exposed to alachlor residues from agricultural and nonagricultural uses. These exposures were discussed in detail in Chapter II of this document.

1. Risks from Alachlor

Data demonstrate that administration of alachlor to laboratory animals induced statistically and biologically significant oncogenic responses at multiple sites at multiple doses in two sexes of one rodent species (rat) and at one site at one dose in one sex of a second species (mice). Mutagenicity studies indicate that alachlor is weakly genotoxic. Alachlor is also expected to be rapidly metabolized and eliminated based on a rat metabolism study. The Agency has concluded that alachlor is a Class B2 carcinogen. The specific risk estimates for oncogenicity are presented in Chapter II and include dietary, applicator, and drinking water risks to humans from exposure to alachlor residues. The potential risks from dietary exposure to alachlor are 2×10^{-5} (assuming 100 percent crop treated) and 1×10^{-6} (assuming actual percent crop treated). The potential risks to applicators range from 4×10^{-5} to 4×10^{-7}.

2. Risks from Chemical Alternatives to Alachlor

Table 19 summarizes the use patterns for the most likely alternatives to alachlor. Based on the current and projected use patterns of those compounds, the primary alternative appears to be metolachlor.

Metolachlor has a fairly complete toxicology data base, lacking only mutagenicity studies. A Registration Standard was issued in 1980. Four studies examining the oncogenic potential of metolachlor have been submitted to the Agency. Two of these studies were in the Sprague-Dawley strain of rat and were found to be positive for the induction of neoplastic nodules in the liver at the highest tested dose level of 3000 ppm. The National Academy of Sciences (1980) has concluded that "the neoplastic nodule is a manifestation of the process of hepatocarcinogenesis . . . . It is induced by a variety of hepatocarcinogens but not by noncarcinogenic agents." However, two mouse studies of metolachlor are considered to be negative with respect to oncogenic potential. The potential of metolachlor to cause cancer in laboratory animals, therefore, is substantially less clear than the weight of evidence on the carcinogenic potential of alachlor.

Based on the chronic rat study, the oncogenic potency of metolachlor (as indicated by the Q_1^* of 2.1×10^{-3}) appears to be much less than that of alachlor ($Q_1^* = 1 \times 10^{-1}$). However, it is noted that the testing of these two compounds was conducted by different laboratories and used different strains of rats. The use patterns, dietary Theoretical Maximum Residue Contributions (TMRC), applicator exposure patterns and potential for drinking water contamination appear to be similar for the two compounds.

C. Benefit Conclusions

The benefits of alachlor were assessed in terms of economic impacts which would result if registrations for its specific uses were cancelled and if its users employed available alternatives. The analysis describes the benefits which would be foregone to users and others affected if alachlor were cancelled for use on each of its registered use sites. Such an assessment provides a baseline estimate of the value of alachlor as currently used and registered to the agricultural community. It also provides a worst case assessment of the economic impact if all alachlor users were to switch to alternative pest control measures as a result of the Agency's proposed regulatory action, an outcome the Agency believes is extremely remote. On the contrary, as indicated in the discussion below, the Agency believes that its proposed regulatory action will have minimal impacts on the use and value of alachlor, and that the loss to the agricultural community would be significantly less than if alachlor were unavailable.

Table 19. Toxicity Data Base for Alternatives to Alachlor

	Pivotal Data	Data Gaps	Basis for Acceptable Daily Intake Calculation
Metolachlor	- 6 month dog feeding, NOEL = 100 ppm - 2-Year supplementary (IBT)[1] onco. in rat: liver tumors - 2-Year chronic in rat (repeat study): liver tumors, NOEL = 30 ppm (testicular atrophy) - 2-Year onco. in mouse negative at 3000 ppm (Highest Dose Tested [HDT][2] Industrial Bio-Test, validated) - 3-generation rat reproduction = 300 ppm - Teratology rat NOEL = 360 mg/kg (HDT) - Teratology rabbit NOEL = 360 mg/kg - Mutagenicity: negative (2 tests) - Positive skin sensitizer - 2-Year onco. in mouse: negative at 3000 ppm (HDT)	Additional mutagenicity	6-month dog feeding, NOEL = 100 ppm, liver tumors $Q_1^* = 2.1 \times 10^{-3}$
Vernolate	- 2-Year mouse feeding/onco. negative NOEL $\geq$ 100 mg/kg/day (HDT)	All other pivotal data: chronic, onco., terato and reproduction	90-day rat feeding, not Core classified*: NOEL = 640 ppm
Pendimethalin	- Rabbit teratology, NOEL = 60 mg/kg (HDT) - 2-Year dog feeding, NOEL = 12.5 mg/kg/day (hepatic toxicity) - Mutagenicity: negative (2 tests)	All other pivotal data	90-day rat feeding, not Core classified: NOEL = 500 ppm

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* The core classification system was developed by the Office of Pesticide Programs in 1977 to assess the adequacy of toxicology studies.

[1] IBT = Industrial Bio-test Laboratories

[2] HDT = Highest Dose Tested

Table 19. Toxicity Data Base for Alternatives to Alachlor (cont'd)

	Pivotal Data	Data Gaps	Basis for Acceptable Daily Intake Calculation
Metribuzin	- Rabbit teratology; NOEL = 15 mg/kg/ (HDT) - Mutagenicity: negative (3 tests) All the other pivotal data were not Core classified: - 2-Year dog feeding, NOEL = 100 ppm - 2-Year rat feeding/onco., negative for oncogenicity; NOEL = 300 ppm - 2-Year mouse onco. negative at 3200 ppm (HDT) - 3-generation reproduction, NOEL = 300 ppm - Rat teratology, NOEL = 100 mg/kg (HDT)	Several pivotal studies were not Core classified and will be reevaluated in the Registration Standard.	Two-year dog feeding, not Core classified: NOEL = 100 ppm
Oryzalin	- 3-generation reproduction NOEL = 250 ppm (LDT) - Rat teratology NOEL = 225 ppm (HDT) - Rabbit teratology NOEL = 125 mg/kg (HDT) - Chronic feeding/onco. in mouse NOEL = 500 ppm, not oncogenic at 3650 ppm (HDT) - 2-Year rat feeding/onco.: NOEL = 300 ppm, potential oncogen: skin tumors. - Mutagenicity: negative (3 studies)	One mutagenicity study: dominant lethal	90-day rat feeding not Core classified NOEL = 750 ppm; Potential oncogen in rat: skin tumors $Q_1^* = 3.375 \times 10^{-2}$

Table 19. Toxicity Data Base for Alternatives to Alachlor (cont'd)

	Pivotal Data	Data Gaps	Basis for Acceptable Daily Intake Calculation
Trifluralin	- Rabbit teratology, NOEL = 450 mg/kg (Supplementary Data) - 4-generation rat reproduction, NOEL = 200 ppm (LDT) (Supplementary Data) All other pivotal data were not Core classified. - Mouse onco: positive, hepatocellular adenoma and carcinoma, alveolar-bronchiolar adenoma, and squamous-cell carcinoma in female mice. - Rat onco: negative at 6500 ppm (HDT) - Reproduction - dog, NOEL = 25 mg/kg - 2-Year feeding rat, NOEL = 2000 ppm - 3-Year dog, NOEL = 10 mg/kg (liver weight changes) - 2-Year dog, NOEL = 10 mg/kg (HDT)	Pending reevaluation of pivotal data	90-day dog feeding, not Core classified: NOEL = 400 ppm
Propachlor	- Rat teratology, NOEL = 200 ppm (HDT). All the other pivotal data are not adequate to regulate this chemical: invalid or unvalidated IBT studies.	IBT replacement studies for all subchronic, chronic, and teratology data base.	90-day rat feeding, unvalidated IBT study with a NOEL = 266 ppm
Diphenamide	Pivotal data not Core classified: - 3-generation reproduction, NOEL = 10 mg/kg (fetotoxicity) - 2-Year rat feeding, NOEL = 10 mg/kg - 2-Year rat feeding, NOEL = 3 mg/kg	Pending reevaluation of pivotal data. Also no teratology study was initially submitted.	2-Year dog feeding, not Core classified: NOEL = 3 mg/kg

Table 19. Toxicity Data Base for Alternatives to Alachlor (cont'd)

	Pivotal Data	Data Gaps	Basis for Acceptable Daily Intake Calculation
Atrazine	- Rat teratology, NOEL = 100 mg/kg - Negative mutagenicity (1 study). The other pivotal data are not adequate (Core supplementary) to regulate this chemical. - 2-Year dog feeding, NOEL = 150 ppm (LDT) - 3-generation rat reproduction, NOEL = 100 ppm (HDT) using the 80W formulation	Extensive data gaps: All chronic, teratology, reproduction and additional mutagenicity.	2-Year dog feeding, Core supplementary: NOEL = 150 ppm
Simazine	- 3-generation reproduction NOEL $\geq$ 100 ppm (only one dose tested) - A questionable 2-year chronic feeding/onco. study in rat needs to be repeated (NOEL $\geq$ 100 ppm [HDT] using a 50% ai product)	Extensive data gaps: All chronic and teratology. A questionable onco. in rat needs to be replaced.	Apparently an Invalid study (unidentified at the present time) was used for ADI calculation.
Cyanazine	- teratology in Fisher rat: a potential weak teratogen, (NOEL for study not yet determined: 10 mg/kg for microophthalmia/anophthalmia, and pending additional studies, the NOEL may be lower than 1 mg/kg (LDT) for liver induced hernia) - teratology in SD rat: negative at 30 mg/kg (HDT), (however, MTD not tested). - teratology in rabbit: negative at 4 mg/kg (HDT), NOEL = 1 mg/kg/day - 2-Year onco. in mice: negative at 1000 ppm (HDT)	All other pivotal data: chronic in 2 sp., onco. in rat, 3-gen. reproduction and additional mutagenicity studies	2-Year rat feeding, Core supplementary: NOEL = 12 ppm

As detailed in Chapter III, should alachlor be cancelled, losses would be relatively small compared to the overall affected crops. Cancellation of all registrations alachlor would be expected to result in first year losses at the farm level of $510 to $758 million, which represents both increased costs of weed control and decreased value of production.

Metolachlor is the principal alternative which currently costs slightly more than alachlor on a per pound active ingredient basis but generally costs slightly less on a per acre treated basis. Metolachlor will not attain the same yield in all cases, as discussed in more detail in Chapter III.

It is projected that the burden of the loss of alachlor would largely be borne by the farmer and about one-third of the burden would be shifted to the consumer. The overall benefits loss to society would range from about $302 to $508 million the first year. Such overall losses would be nominal to consumers of food products affected.

D. Development of Regulatory Options

There are three basic options for the regulation of all pesticides:

Option 1 - Continuation of Registration without Changes

Option 2 - Continuation of Registration with Modifications to the Terms and Conditions of Registration

Option 3 - Cancellation of Registration

The two extreme options, Option 1, Continuation of Registration without Change and Option 3, Cancellation of Registration, are at the opposite ends of the risk/benefit spectrum. Adoption of Option 1 would be appropriate when the Agency has concluded that the level of risk is reasonable in light of the pesticide's benefits and that further risk reduction measures are not necessary to assure that the use of the pesticide meets the statutory standard for continued registration.

Adoption of Option 3, cancellation, would be appropriate when the Agency has concluded that the risks from a use outweigh the benefits of that use, and that these risks cannot be mitigated to a reasonable level, in light of the benefits, by any other measures short of cancellation. Cancellation may affect all uses of a compound, only specific uses or specific formulations, or specific application methods.

Option 2 is appropriate when the risks of a pesticide use can be reduced to a level where the benefits of use outweigh the risks. This risk reduction is accomplished by modifying the terms and conditions of the pesticide's registration. These modifications, which are expressed through the pesticide's labeling are, for the most part, changes in the way the pesticide is used. These changes are designed to reduce exposure to the pesticide and thereby reduce or eliminate the risk from the pesticide. In the discussion that follows, risk reduction measures were considered and evaluated for their potential effectiveness and feasibility.

1. Measures to Reduce Dietary Exposures

a. Cancel Use on Peas

The overall U.S. dietary exposure to alachlor residues would be reduced by 9 percent if alachlor were cancelled for use on peas. This estimate was calculated considering the percent crop treated and estimating the amount of the HEEA metabolite residues in peas. For peas, the Agency has residue data for alachlor and its DEA metabolites. The Agency estimated the amount of HEEA metabolites based on levels found in related leguminous crops. If registrations for use on peas are cancelled there would be little impact to the farmer. Residue data for processed/cooked commodities are not available. The Agency, under the authority of FIFRA section 3(c)(2)(B), has required that processing and cooking studies be conducted to determine the level of alachlor residues in processed peas. These data are due to be received in July 1987 and will be considered when the Agency issues its final determination.

b. Cancel Use on Dry Beans

The U.S. dietary exposure would be reduced by 32 percent for dry beans if alachlor registrations for use on this crop were cancelled. This estimate was calculated considering percent crop treated and estimating the amount of the HEEA metabolite residues in dry beans. For dry beans, the Agency has residue data for alachlor and its DEA metabolites. The Agency estimated the amount of HEEA metabolites based on levels found in related leguminous crops. Overall impact to farmers if use on dry beans is cancelled is estimated to be $0.6 to $1.6 million (about 25 percent of the net returns for affected farmers). Impact is localized to Nebraska, Colorado, and southern Michigan. Residue data for processed/cooked commodities are not available. The Agency, under the authority of FIFRA section 3(c)(2)(B), required that processing and cooking studies be conducted to determine the level of alachlor residues in processed dry beans. These data are due to be received in July 1987 and will be considered when the Agency issues its final determination.

c. Cancel Use on Lima Beans

The U.S. dietary exposure would be reduced by 7 percent for lima beans if alachlor registrations for use on this crop were cancelled. This estimate was calculated considering percent crop treated and estimating the amount of the HEEA metabolite residues in lima beans. For lima beans, the Agency has residue data for alachlor and its DEA metabolites. The Agency estimated the amount of HEEA metabolites based on levels found in related leguminous crops. If use on lima beans is cancelled, the impact to farmers would be small based on the Agency's limited data base on this crop. Residue data for processed/cooked commodities are not available. The Agency, under the authority of FIFRA section 3(c)(2)(B), required that processing and cooking studies be conducted to determine the level of alachlor residues in processed dry beans. These data are due to be received in July 1987 and will be considered when the Agency issues its final determination.

d. Lower Application Rates

Generally, application rates could not be reduced on any crop without affecting the efficacy of alachlor on target weeds. On soybeans and peanuts, the number of applications can be limited to one per year instead of the two applications which are currently on the label. However, this will not change the Agency's U.S. dietary exposure estimates because the dietary risk calculations assume only one application per year for these crops, which is the common practice.

2. Measures to Reduce Exposure to Applicators, Mixer/Loaders, and Field Workers

a. Label Alachlor as a Restricted Use Pesticide

This will ensure that all farmers who use alachlor will be trained in its proper use or directly supervised by a certified applicator.

Approximately 65 percent of all commercial and private farmers who use alachlor are already certified under state certification programs, according to data provided by the registrant. The Agency expects this percentage to be much higher due to EPA's recent requirement that users of granular formulations used to control corn rootworm must be certified.

Private farmers would comprise the majority of the remaining uncertified group. The cost to each farmer of obtaining certification would be minimal; some states do not charge a fee to private farmers for certification. The costs of additional certification and training attributable to this proposed measure would be borne by the states. EPA estimates that the states spend between $4 and $5 million annually on certification and training programs. The incremental cost attributable to the requirement that alachlor users be certified would be minimal.

Commercial applicators who use alachlor are most likely already certified due to their usage of other restricted use pesticides, and would incur no additional costs under this proposal.

As a result of the reclassification of alachlor, some decline in the usage of alachlor may occur. However, the decline is not expected to be significant.

b. Require Use of a Closed Mixing/Loading System - (Equal to or substantially similar to the Monsanto shuttle system)

The shuttle system is a 200-gallon reusable tank with a pump which was developed by Monsanto Chemical Company to reduce applicator exposure to their agricultural chemicals. The shuttle system is a type of closed loading system. A closed system is defined as any method that eliminates open atmosphere contact with a pesticide or its rinse solution (if any) during transfer from the original container to the application equipment or system. Use of a closed loading system reduces mixer/loader exposure by up to fortyfold. The shuttle system is currently economically feasible only when 300 or more acres are going to be treated.

This requirement would affect approximately 40 percent of the farms using alachlor. The Agency assumes that most of these large farms already use closed mixing/loading systems, such as the shuttle system developed by Monsanto, and therefore expects this requirement to have a minimal economic impact.

c. Require Use of Enclosed Cabs

An enclosed cab is any vehicle or aircraft with a nonporous covering that totally surrounds the operator of the vehicle. The enclosed cab could greatly reduce applicator exposure over that received in non-enclosed cabs or enclosed tractor cabs with open windows.

d. Reinstate Aerial Application on Label

Prior to the issuance of the PD-1, aerial application was voluntarily removed from the alachlor label by the registrant. In response to rebuttals to the PD-1, the Agency evaluated published data showing that pilots receive less exposure than ground boom applicators. The Agency concludes that exposure to pilots is less than for ground applicators. This option would reinstate aerial application on the labels of alachlor products.

3. Measures to Reduce Drinking Water Exposures

a. Surface Water

(1) Require the Banding of Treated Areas

Under banding, applicators would apply alachlor to a continuous restricted area such as on or along a crop row rather than over the entire field area. Banding is only applicable to fields that are conventionally tilled. Banding would reduce the amount of alachlor applied to a given field by 1/2 to 2/3. However, the trend for farmers is to use reduced till instead of conventional till in order to prevent soil erosion.

(2) Require Soil Incorporation

Alachlor would be applied and tilled into the top one or two inches of soil before seeding or transplanting. Soil incorporation is expected to reduce alachlor contamination of surface water by an unknown amount. Soil incorporation can only be used on fields that are conventionally tilled. The agricultural community is changing from conventional tillage to reduced till to prevent soil erosion.

(3) Impose Restrictions on Community Water Authorities

Community water authorities would be required to blend ground and surface water supplies, close intake pipes during runoff periods, or use granular activated carbon or powdered activated carbon when alachlor levels exceed 1 ppb. This requirement would be imposed under the Safe Drinking Water Act, not FIFRA.

(4) Establishment of Contaminant Level

A Recommended Maximum Contaminant Level (RMCL) for alachlor of zero has been established by EPA's Office of Drinking Water. The Agency plans to promulgate regulations establishing a Maximum Contaminant Level (MCL) for alachlor under the Safe Drinking Water Act in the near future. These regulations would require the treatment of drinking water which contains alachlor residues in excess of the MCL. This measure would sufficiently reduce risks from drinking water to reasonable levels.

b. Ground Water

(1) Regulate in accordance with the scheme being developed for other pesticides

The Agency is developing a comprehensive strategy for addressing pesticides in ground water. Once this strategy is developed, and sufficient data are available to estimate alachlor's impact on ground water, the Agency would regulate alachlor in accordance with that strategy.

(2) Continued Registration - No Restrictions

Based on available information, the occurrence of alachlor in ground water cannot be estimated. Several ground water and soil leaching studies are currently underway. A decision on whether to allow continued registration based on ground water concerns cannot be made at this time.

E. Risk/Benefit Analysis of Regulatory Options

1. Dietary Exposure

a. Cancel Use on Peas

If the registrations of alachlor products for use on peas were continued without restriction (Option 1), the dietary risks would be reduced by 9 percent. Residues in processed or cooked peas were not estimated.

If the registrations of alachlor products for use on peas were cancelled (Option 3), all risks associated with consuming alachlor-treated peas would be eliminated. The cancellation of alachlor use on peas would result in little impact to farmers.

The Agency has decided to delay the final regulatory decision on alachlor use on peas until residue data on processed and cooked peas are submitted and reviewed. The use on peas will be allowed to continue while these data are being generated and reviewed.

b. Cancel Use on Dry Beans

If the registrations of alachlor products for use on dry beans were continued without restriction (Option 1), the dietary risks would be reduced by 32 percent.

If the registrations of alachlor products for use on dry beans were cancelled (Option 3), all risks associated with consuming alachlor treated dry beans would be eliminated. The cancellation of alachlor use on dry beans would result in little impact to farmers.

The Agency has decided to delay the final regulatory decision on alachlor use on dry beans until residue data on processed beans are submitted and reviewed. The use of alachlor on dry beans will be allowed to continue while the data are being generated and reviewed.

c. Cancel Use on Lima Beans

If the registrations of alachlor products for use on lima beans were continued without restriction (Option 1), the dietary risks would be reduced by 7 percent.

If registrations of alachlor products for use on lima beans were cancelled (Option 3), all risks associated with consuming alachlor treated lima beans would be eliminated. The cancellation of alachlor use on lima beans would result in little impact to farmers based on the limited data available on this crop.

The Agency has decided to delay the final regulatory decision on alachlor use on lima beans until residue data on processed beans are submitted and reviewed. The use of alachlor on lima beans will be allowed to continue while the data are being generated and reviewed.

2. Applicator Exposure

The potential oncogenic risk to persons applying alachlor to crops was estimated as follows:

Applicator	Days Exposed/Year	Lifetime Risk Estimates
Private Farmers	1	4×10^{-5} to 4×10^{-7}
Private Farmers	3	1×10^{-4} to 1×10^{-6}
Commercial Applicators	15	6×10^{-4} to 6×10^{-6}

The Agency has concluded that these risks outweigh the benefits unless they are reduced by modifying the terms and conditions of registration. Therefore, the Agency proposes that alachlor be labeled as a restricted-use pesticide. This will ensure that all farmers, or other applicators, who use alachlor have been trained in its proper use or are supervised by a trained individual. In addition, the Agency proposes the required use of the shuttle or other closed mixing/loading system (a closed system) for individuals treating 300 acres or more annually with alachlor. This requirement would affect large private users of alachlor and commercial applicators and would reduce the upper limit of their risk from 10^{-4} to 10^{-5}. Closed mixing/loading systems are not considered economically feasible or practical, and will not be required for small users who treat less than 300 acres annually of alachlor. This group of farmers already had risk estimates in the 10^{-5} range due to the reduced frequency of their exposure to alachlor.

3. Drinking Water Exposure

a. Surface Water

EPA's Office of Drinking Water has not yet proposed a MCL for alachlor but is expected to do so within the next 6 months. If that level were in the .5 to 1 ppb range, the dietary risk would be 2 x 10^{-6} to 4 x 10^{-6}, depending on the level set. This would sufficiently reduce risks from this source to reasonable levels.

b. Ground Water

The true extent of the occurrence of alachlor in ground water is not known, and cannot be properly estimated for most areas of the country. The Agency's position is that further monitoring will be necessary considering the large volume, multiregional use of alachlor, and the lack of statistically representative data from the cited studies. Several ground water and soil leaching studies are being conducted by the registrant, various states, and the U.S. Department of Agriculture and the U.S. Geological Survey. The risk posed by alachlor from exposure through ground water will continue to be assessed along with the need for any regulatory action as new data become available.

F. Summary of Proposed Decision - All Agricultural and Nonfood Uses

1. Current registrations for the use of alachlor on agricultural and nonfood crops will be allowed to continue provided the following conditions and label modifications are met:

- The application of alachlor is restricted to use by certified applicators or persons under their direct supervision. The alachlor label must be amended to include the following statement:

  Restricted Use

  due to oncogenicity

  "For retail sale to and use only by Certified Applicators or persons under their direct supervision and only for those uses covered by the Certified Applicator's certification."

- The following tumor warning statement imposed in the alachlor Registration Standard must remain on the label:

  > "The use of this product may be hazardous to your health. This product contains alachlor which has been determined to cause tumors in laboratory animals."

- The use of a closed mixing/loading system is required to be used by all mixer/loaders and/or applicators who treat 300 acres or more annually.

- Aerial application will be allowed to be reinstated on the alachlor label with the following additional label restriction:

  > "Human flaggers prohibited. Aerial application may be performed using mechanical flaggers ONLY."

2. The risk posed by alachlor from exposure through ground water will continue to be assessed along with the need for any regulatory action as new data become available.

3. The Agency's final regulatory decision on registrations of alachlor for use on peas, dry beans, and lima beans is being deferred, pending receipt of required residue data for each of these commodities when they are processed. These data are required to be submitted pursuant to a 3(c)(2)(B) Notice issued to the registrant in June 1986. These data are due to be submitted by July 1987.

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APPENDIX A

Table A. Alachlor in Surface Water

State	Sampling Site	# Positive / Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc. (ppb)	Sampling Period
OH (1981) (Baker, et al. 1981)	Honey Creek, Melmore	121/158	57.0	06/09	5.48	03/30/81-09/07/81
	Honey Creek, Tiro	4/5	3.87	06/09	1.13	06/09/81-09/07/81
	Upper Honey Creek	42/46	75.6	06/09	10.08	05/04/81-09/07/81
	Bean Creek, Powers	8/12	32.7	06/09	3.79	03/31/81-07/07/81
	Ottawa River, Allentown	5/10	14.2	06/16	3.18	03/31/81-07/07/81
	Rocky River, Berea	1/5	0.035	05/20	0.01	03/31/81-05/20/81
	Sandusky River, Tindell (Fremont)	16/17	42.5	06/11	10.29	05/26/81-09/07/81
	Sandusky River, Mexico	2/2	0.455	09/07	0.30	06/04/81-09/07/81
	Sandusky River, Defiance	28/30	104.6	06/08	10.94	05/29/81-07/06/81
	Wolf Creek, East Br	3/3	67.7	06/09	29.26	06/09/81-06/14/81
	Wolf Creek, West Br	3/3	54.1	06/09	27.74	06/09/81-06/14/81
	Maumee River, Waterville	1/1	6.97	06/16	-	06/16/81
OH (1982) (Baker, 1983b)	Maumee River, Bowling Green (6,313 mi^2)	45/53	9.27	05/28	1.88	12/08/81-08/23/82
	Sandusky River, Fremont (1,251 mi^2)	45/51	18.19	05/29	4.16	12/07/81-08/23/82
	Honey Creek, Melmore (149 mi^2)	56/65	75.0	05/24	7.99	12/07/81-08/23/82
	Lost Creek, Defiance (4.4 mi^2)	41/51	18.46	05/22	0.83	12/08/81-08/23/82
	River Raisin, Monroe, MI (1042 mi^2)	21/25	8.16	05/29	1.71	04/05/82-08/28/82
	Cuyahoga River, Independence (707 mi^2)	8/24	0.60	07/19	0.07	12/07/81-08/23/82

Table A. Alachlor in Surface Water (cont'd)

State	Sampling Site	# Positive / Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc. (ppb)[1]	Sampling Period
OH (1983) (Baker, 1986a)	Maumee River, Bowling Green	42/62	7.49	07/01	1.97 (0.45,1.47)[2]	04/04/83-09/26/83
	Sandusky River Fremont	36/58	4.92	06/28	0.78 (0.25,0.76)[2]	04/04/83-09/26/83
	Honey Creek, Melmore	47/68	8.87	05/31	1.91 (0.66,1.60)[2]	04/15/83-09/26/83
	Upper Honey Creek, New Washington	17/58	8.69	06/07	0.69	04/12/83-09/27/83
	Rock Creek, Tiffin	21/46	11.89	06/29	1.39	04/12/83-09/20/83
	Lost Creek, Defiance	25/51	34.44	06/06	3.55	04/04/83-09/26/83
	River Raisin, near Monroe MI	11/32	7.52	06/30	0.73	04/09/83-10/02/83
	Cuyahoga River, Independence	1/25	1.16	07/11	0.04	04/11/83-09/26/83
OH (1984) (Baker, 1986b)	Maumee River, Bowling Green	52/88	18.35	05/24	2.59 (0.74,1.95)[2]	04/02/84-10/22/84
	Sandusky River, Fremont	50/79	9.10	06/28	1.56 (0.53,1.50)[2]	04/02/84-10/29/84
	Honey Creek, Melmore	70/100	22.89	07/07	2.05 (0.88,2.01)[2]	04/02/84-10/29/84
	Upper Honey Creek, New Washington	14/32	0.85	05/30	0.16	05/30/84-10/23/84
	Rock Creek, Tiffin	40/87	7.42	05/21	0.78	04/01/84-10/23/84
	Lost Creek, Defiance	35/57	33.11	05/21	3.14	04/02/84-10/29/84
	River Raisin, near Monroe MI	21/43	5.03	05/27	0.43	04/08/84-10/21/84
	Cuyahoga River, Independence	5/20	0.35	07/02	0.05	04/24/84-09/10/84
OH (1985) (Baker, 1985)	Maumee River, Bowling Green	23/56	3.61	06/11	0.43 (0.20,0.49)[2]	02/18/85-09/16/85
	Sandusky River, Freemont	57/82	16.97	05/21	2.85 (0.78,2.44)[2]	02/11/85-09/23/85
	Honey Creek, Melmore	87/121	17.32	05/19	3.00 (0.91,2.13)[2]	02/11/85-09/23/85
	Upper Honey Creek, New Washington	87/125	1.65	05/29	0.51	02/11/85-09/17/85
	Rock Creek, Tiffin	71/118	12.92	05/19	2.53	02/12/85-09/24/85
	Lost Creek, Defiance	18/63	1.03	06/03	0.43	02/18/85/09/23/85
	River Raisin, near Monroe MI	12/31	5.61	05/28	1.52	02/24/85-09/22/85
	Cuyahoga River, Independence	4/29	0.30	03/25	0.244	03/11/85-09/10/85

Table A. Alachlor in Surface Water (cont'd)

State	Sampling Site	# Positive/Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc. (ppb)	Sampling Period
(Miltner, 1984)	Sandusky River, Tiffin	-/17	13.7	06/27	5.4	05/21/84-07/09/84
	Sandusky River, Fremont	-/7	5.5	05/21	3.4	05/18/84-06/25/84
	Maumee River, Bowling Green	-/20	12.2	05/25	3.6	05/15/84-07/16/84
OH (Columbus) (Binder, 1984: Raw Water Intake)	Dublin Road Water Plant (raw)	8/8	5.71	06/30	1.19	06/30/81-01/11/82
	Dublin Road Water Plant (raw)	11/11	6.10	06/11	3.18	06/10/82-07/21/82
	Dublin Road Water Plant (raw)	7/7	3.65	07/14	3.23	07/14/83-08/16/83
	Dublin Road Water Plant					
	DRWP Intake	4/4	0.45	01/--	0.26	10/81-01/82
	Griggs Reservoir	4/4	0.48	01/--	0.31	10/81-01/82
	Griggs Reservoir	3/3	5.76	06/26	2.14	06/26/83-08/29/83
	O'Shaughnessey Reservoir	4/4	0.51	01/--	0.37	10/81-01/82
	O'Shaughnessey	3/3	5.07	06/26	1.84	02/12/83-08/29/83
	Morse Road Water Plant					
	MRWP Intake	4/4	0.25	10/--	0.20	10/81-01/82
	Alum Creek Reservoir	4/4	0.52	10/--	0.42	10/82/01/82
	Alum Creek Reservoir	3/3	0.95	06/26	0.79	02/12/83-08/29/83
	Hoover Reservoir	4/4	0.40	10/--	0.35	10/81-01/82
	Hoover Reservoir	3/3	0.41	08/--	0.35	02/12/83-08/29/83
	Other Samples					
	Bokes Creek	1/1	7.83	06/22	-	06/22/83
	O'Shaughnessey Top	1/1	4.05	06/27	-	06/27/83
	O'Shaughnessey Bottom	1/1	6.33	06/27	-	06/27/83
	O'Shaughnessey LLYC	1/1	3.57	06/26	-	06/26/83

Table A. Alachlor in Surface Water (cont'd)

State	Sampling Site	# Positive Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc. (ppb)	Sampling Period
IA (Johnson et al., 1982,1984)	Four Mile Creek Watershed Site 4	27/145	96.0	05/29	1.77	05/02/76-09/19/76
		4/86	0.3	05/22	0.01	04/10/77-10/01/77
		45/130	54.5	06/19	2.32	05/01/78-09/14/78
		205/222	267.7	06/04	5.55	05/01/79-10/29/79
		101/104	208.7	06/15	20.27	05/08/80-08/11/80
Clayton County (Hallberg) et al., 1983)	Big Spring (surface water)	9/9	20.0	06/08	2.86	02/24/82-12/29/82
IA (Splinter, 1986)	Cedar River, Cedar Rapids	-	9.1	06/--/81	-	02/75-07/82
	Iowa River, Iowa City	-	9.0	06/--/82	-	02/75-07/82
	Little Sioux River, Cherokee	-	0.92	05/--/82	-	02/75-07/82
	Mississippi River, Dubuque	-	3.6	06/--/80	-	02/75-07/82
	Mississippi River, Keokok	-	9.5	01/--/77	-	02/75-07/82
	Mississippi River, Lounere Bluffs	-	1.9	05/--/82	-	02/75-07/82
	Raccoon River, Des Moines	-	1.2	06/--/79	-	02/75-07/82
	Shank River, Oskaloosa	-	1.9	06/--/80	-	02/75-07/82
	Upper Iowa River, Decorah	-	18.0	06/--/81	-	02/75-07/82
	Nishna Botna River, Hamburg	-	8.1	06/--/80	-	02/75-07/82

Table A. Alachlor in Surface Water (cont'd)

State	Sampling Site	# Positive / Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc.[1] (ppb)	Sampling Period
MI (1985) (Baker, 1985)	Black River, Fargo, MI	18/37	4.2	06/02	1.03	04/30/08-08/09/85
	Clinton River, Mt. Clemens, MI	9/39	2.45	06/01	0.72	04/30/85-08/09/85
	Belle River, St. Clair, MI	5/33	1.41	06/01	1.23	04/30/85-08/08/85
	Pine River, St. Clair, MI	0/37	-	-	-	04/30/85-08/08/85
KS (Butler, 1985)	Amb. Stream WQN	50/-	-	-	-	1977-1984
	Federal Lakes	-/21	3.1	-	0.82	1974-1984
	Small Lakes	-/1	0.36	-	0.36	1979-1984
Canada (Bradley, 1985)	Dresden (in SW Region) (Raw Water)	13/36	9.0	06/01	0.62	05/28/85-08/13/85
	Southwestern Region (exc. Dresden)					
	Raw Water	17/289	Trace[3]	-	-	05/02/85-08/85
	Ambient	17/231	51.0			05/14/85-08/85
	West Central Region (Raw Water)	6/137	Trace[3]	-	-	05/02/85-08/85
	South Eastern Region (Raw Water)	2/62	Trace[3]	-	-	05/02/85-08/85
Canada (Frank et al., 1982)	Ontario (11 agricultural watersheds)	-	9.0		0.02	1975-1976
		-	ND[4]	-	ND[4]	1976-1977

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[1] The mean concentration is a simple arithmetic mean of all samples analyzed. A value of 0.00 was assigned to samples where alachlor was not detected.

[2] Time weighted average = $c_{twa} = \frac{c_i t_i}{t_i}$, where c_i is the concentration in i^{th} sample and t_i is the duration of the time the i^{th} sample characterizes in the stream system. First number calculated with no limit to time factor and second number using a 1-day maximum time factor.

[3] Trace = positive sample with concentration less than 1 ppb, but greater than 0.5 ppb

[4] ND = not detected

Table B. Summary of Alachlor Monitoring Data Extracted from STORET
(All Sites reporting ≥ 2 ug/L (2 ppb)

State	Sampling Site	# of Samples ≥ 2 ppb	Reporting Dates for ≥ 2 ppb	Range of Conc. (ppb)	Date of Peak Conc.
IA	Chariton River SSE of Russell	7	06/07-07/28/78	3.0-101.0	07/19/78
	SPRK Chariton R. N.W./Promise City	2	06/28-07/05/78	2.4-14.0	06/28/78
	Iowa River Near Marengo	1	05/29/84	6.50	05/29/84
	Shell Rock River Near Northwood	1	06/18/84	6.80	06/18/84
	Cedar River at Cedar Falls	2	06/10-06/20/84	8.2-22.0	06/10/78
	Cedar River at Gilbertville, Blw waterloo	2	06/10-06/20/84	4.9-17.0	06/10/84
	Cedar R. NR Bertram	4	05/17-06/21/84	2.7-6.6	06/11/84
	Cedar River at Floyd	2	06/09-06/19/84	7.5-21.0	06/09/84
	Cedar River Near Carville	2	06/10-06/19/84	9.0-22.0	06/10/84
	Salt Creek NR Elboron	1	05/29/84	4.2	05/29/84
	Big Bear Creek at Laddra	1	05/29/84	2.3	05/29/84
	Iowa River near Rowan	1	05/22/85	2.8	05/22/85
	Iowa River at Iowa City	1	05/23/85	2.1	05/23/85
	Walnut R. NR Eldorado Butler	1	12/19/78	2.4	12/19/78
KS	Missouri River at Kansas City Wyandotte	1	06/22/78	2.0(K)	06/22/78
	Wakarasa River below Clinton Dam, Douglas	1	05/07/78	3.2	05/07/78
	Kansas River at Manhattan Riley	1	06/22/78	50.0(K)	06/22/78
	Topeka, Whitelake, Shawnee	1	08/11/81	2.0(K)	08/11/81
	Overland Park, Southpark Lake, Johnson	1	08/12/81	2.0(K)	08/12/81
	Mamaton R. NR Ft. Scott Burbon	1	07/07/82	4.2	07/07/82
	Soldier Creek near Delta, Shawnee	1	07/13/82	4.4	07/13/82
	Big Blue River near Oketo, Marshall	1	06/16/83	3.2	06/16/83
	Big Blue River near Oketo, Marshall	1	06/22/83	2.5	06/22/83
	Big Blue River near Blue Rapids, Marshall	1	06/22/83	2.1	06/22/83
	Big Blue R. Bl. Tuttle Cr. Res. Riley	1	06/12/84	2.4	06/12/84
	Big Blue River Hwy. 9 ans 27 Bridge, Blue Rapids	1	05/24/85	2.6	05/24/85
	Wolf R. near Sparks Doniphan	1	05/25/84	3.4	05/25/84
	Tuttle Cr. Lake 10.6 mi uplake/dam Pottawatomic	1	06/10/85	3.1	06/10/85
	Black Vermillton in S. Frankfort, Marshall	1	05/14/85	8.1	05/14/85

Table B. Summary of Alachlor Monitoring Data Extracted from STORET
(All Sites reporting $\geq$ 2 ug/L (2 ppb) (cont'd)

State	Sampling Site	# of Samples $\geq$ 2 ppb	Reporting Dates for $\geq$ 2 ppb	Range of Conc. (ppb)	Date of Peak Conc.
OH	Maumee River at Waterville Lucas	15	05/24-06/20/82	2.8-8.9	05/28/82
	2.3 miles upstream from Ballville Dam, Sandusky River	18	05/24-07/02/82	2.0-18.2	05/09/82
	Bridge State Rts 100 and 67 Seneca, Honey Creek	22	05/23-06/30/82	2.6-69.6	05/25/82
MI	River Raisin near Monroe	5	05/28-06/01/82	4.2-8.2	05/28/82
VA	Blackwater Creek Virginia Beach	1	05/23/79	3.4	05/23/79
	West Conewago Creek, 0.1 mile above mouth, York	2	06/04-07/09/81	3.8-11.0	06/04/81
PA	Codorus Creek, 0.5 miles above mouth, York	3	06/04-09/16/81	2.0-6.1	06/04/81
	Codorus Creek, below Springettsburg, STP, 4.7 miles A.B.M. York	2	06/04-09/07/81	2.6-2.8	06/04/81
	Little Conestoga Creek near Churchtown, Lancaster	3	05/21-05/22/85	30.0-60.0	05/21/85
	Little Conestoga Creek, site 3A near Morgantown Lancaster	1	05/03/85	3.5	05/03/85
MD	Octoraro Creek, Br at Rowlansville cectl	1	06/04/81	27.7	06/04/81
NE	1 mile South and 5 1/2 West of Pickrell, Gage	1	05/14/85	10.1	05/14/85
	Pat Creek near Belmont, Lafayette	1	06/15/82	5.5	06/15/82
WI	Maden Branch near Meekers Grove, Lafayette	1	06/15/82	16.0	06/15/82
	Madden Branch - Tributary near Belmont, Lafayette	1	06/15/82	3.2	06/15/82
	Apple River near Shullsburg, Lafayette	1	06/15/82	9.0	06/15/82
	Little River near Manila, Mississippi	1	06/09/76	2.1	06/09/76
AR	Crooked Creek below Harnson, Boone	1	09/24/85	3.0	09/24/85

Table B. Summary of Alachlor Monitoring Data Extracted from STORET (All Sites reporting ≥ 2 ug/L (2 ppb) (cont'd)

State	Sampling Site	# of Samples ≥ 2 ppb	Reporting Dates for ≥ 2 ppb	Range of Conc. (ppb)	Date of Peak Conc.
GA	0.1 mile west of JC GA 305/GS24; GO SO .05 mi, Burke	1	11/28/84	3.0	11/28/84
	1.05 mile south of Jet GA 17/USI (In Wren); E Past, W.I., Jefferson	1	11/28/84	3.0	11/28/84
	0.3 mile east of Hinest Smith on Maple/Sandersville Washington	1	11/28/84	3.0	11/28/84
	Near WTR Tower SW of Jet 175/US 341; Houston	1	12/18/84	3.0	12/18/84
	7.5 miles SE of Jet GA 80/GA 56 Supr at plant Vogtle, Burke	1	11/27/84	3.0	11/27/84
	Under Millen Water Tower/ just E of GA 21, Jenkins	1	11/28/84	3.0	11/28/84
	Located behind Girar Elem. Sch. 1.4 miles NW of Girard Burke	1	11/28/84	3.0	11/28/84
	Adjacent to Marshallville City Hall, Macon	1	12/18/84	3.0(U)	12/18/84
	Oniog S. Briggs St. and Soax St. Valdosta; Lowndes	1	03/28/85	3.0	03/28/85

(K) = Actual value is known to be less than value given.
(U) = Indicates material was analyzed for but not detected.

Table C. Alachlor in Tap Water

State	Sampling Site	# Positive / Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc.[1] (ppb)	Sampling Period
OH						
(Baker et al., 1981)	Tiffin	6/6	14.3	06/17	5.10	06/04/81-09/01/81
(Baker, 1983b)	Tiffin	3/5	11.4	05/28	2.45	12/10/81-05/28/82
(Baker, 1983a)	Tiffin	6/6	14.0	05/25	4.14	05/10/82-07/07/82
(Baker, 1983a)	Bowling Green	-/16	5.91	--	1.87	05/28/83-07/27/83
(Baker, 1983a)	Fremont	-/15	0.47	--	0.22	05/28/83-07/27/83
(Baker, 1983a)	Tiffin	-/15	2.73	--	1.08	05/28/83-07/27/83
(Baker, 1986b)	Bowling Green	51/66	12.25	05/25	2.30	05/15/84-07/16/84
(Baker, 1986b)	Fremont	42/47	10.51	06/27	2.45	05/18/84-08/27/84
(Baker, 1986b)	Tiffin	48/48	13.65	06/27	3.58	05/15/84-07/18/84
Columbus (Bihder, 1984)	Dublin Road Water Plant (tap)	6/6	1.10	08/03	0.47	06/30/81-01/11/82
	Dublin Road Water Plant (tap)	11/11	5.71	06/11	2.82	06/01/82-07/21/82
	Dublin Road Water Plant (tap)	7/7	3.57	07/14	2.39	07/14/83-08/16/83
LA (Nat. Res., 1977)	New Orleans City	-	2.9	1977 (Summer)	-	--

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[1] The mean concentration is a simple arithmetic mean of all samples analyzed. A value of 0.00 was assigned to samples where alachlor was not detected.

Table D. Alachlor in Rainwater

State	Sampling Site	# Positive / Total	Peak Conc. (ppb)	Date of Peak Conc.	Mean Conc.[1] (ppb)	Sampling Period
OH (Baker, 1984)	Tiffin, OH	6/9	1.16	06/13	0.35	06/13/84-07/27/84
(Baker, 1985)		13/24	3.70	05/15	0.62	04/05/85-09/09/85
IN (Baker, 1985)	West Lafayette, Indiana	8/14	1.74	05/16	0.61	05/01/85-09/04/85
WV (Baker, 1985)	Parsons, WV	4/20	0.21	06/12	0.02	04/25/85-08/27/85
NY (Baker, 1985)	Potsdam, NY	5/21	0.34	06/05	0.03	06/05/85-09/09/85

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[1] The mean concentration is a simple arithmetic mean of all samples analyzed. A value of 0.00 was assigned to samples where alachlor was not detected.

Table E. Weekly Maximum and Annual Mean Concentrations of Alachlor in Raw and Finished Surface Water for the 1985 Growing Season

		Alachlor Concentration (ug/L)			
		Weekly Maximum		Annualized Mean	
Location	State	Raw	Finished	Raw	Finished
Bethany	MO	<0.20	<0.20	0 - 0.20	0 - 0.20
Blanchester	OH	1.3	1.1	0.16 - 0.32	0.15 - 0.31
Breese	IL	4.6	4.4	0.29 - 0.44	0.29 - 0.42
Charleston	IL	<0.20	<0.20	0 - 0.20	0 - 0.20
Clarinda	IA	<0.20	<0.20	0 - 0.20	0 - 0.20
Columbus	OH	10.7	10.9	1.3 - 1.5	1.3 - 1.4
Davenport	IA	0.68	0.32	0.02 - 0.21	0.01 - 0.20
Decatur	IL	<0.20	0.29	0 - 0.20	0.03 - 0.20
Greenville	NC	0.26	0.27	0.01 - 0.20	0.01 - 0.20
Kankakee	IL	0.85	0.77	0.09 - 0.24	0.08 - 0.24
Lexington	MO	0.84	0.59	0.05 - 0.23	0.03 - 0.21
Marion	IL	<0.20	<0.20	0 - 0.20	0 - 0.20
Michigan City	IN	<0.20	<0.20	0 - 0.20	0 - 0.20
Monroe	MI	<0.20	<0.20	0 - 0.20	0 - 0.20
Mount Vernon	IN	1.1	1.0	0.06 - 0.24	0.05 - 0.23
Muncie	IN	2.5	2.8	0.26 - 0.40	0.25 - 0.38
Piqua	OH	0.89	0.63	0.05 - 0.23	0.04 - 0.22
Quincy	IL	0.54	0.70	0.04 - 0.21	0.06 - 0.23
Richmond	IN	3.5	3.6	0.57 - 0.68	0.57 - 0.69
Roanoke Rapids	NC	<0.20	<0.20	0 - 0.20	0 - 0.20
Toledo	OH	<0.20	<0.20	0 - 0.20	0 - 0.20
University of Iowa	IA	1.6	1.8	0.10 - 0.28	0.11 - 0.29
Wyaconda	MO	0.24	<0.20	0.02 - 0.20	0 - 0.20
Ypsilanti	MI	<0.20	<0.20	0 - 0.20	0 - 0.20
Overall		10.7	10.9	0.13 - 0.31	0.12 - 0.30

Reference: Monsanto, 1986

*U. S. GOVERNMENT PRINTING OFFICE 1987; 716-002/60561